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In association with

Otto L. Mohr
Professor of Anatomy, Oslo

Tage Kemp
Professor of Human Genetics, Copenhagen

edited by:

Gunnar Dahlberg
Head of the State Institute of Human Genetics and Race Biology, Uppsala

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ÜBER DEN EINFLUSS DER HOMOGAMIE AUF DIE VERTEILUNGSKURVEN DER MENSCHLICHEN KÖRPERHÖHE

Von WIDUKIND LENZ

Die Verteilungskurven der menschlichen Körperhöhe kommen mit ziemlich guter Annäherung dem Typ der Gaußschen Normalkurve gleich. Man kann daraus schließen, daß die ursächlichen Faktoren, welche die Körperhöhe des Menschen bestimmen, zahlreich sind, und daß sie sich etwa nach den Regeln des Zufalls in der Gesamtbevölkerung kombinieren. Auch bei Populationen von Individuen, deren Umweltbedingungen im Wachstumsalter verhältnismäßig ähnlich waren, etwa amerikanischen Studenten, entspricht die Verteilungskurve der Körperhöhe recht gut einer Normalverteilung. Wenn wir annehmen, daß bei derartigen Populationen die Unterschiede der Körperhöhe praktisch ausschließlich durch Unterschiede der Erbanlagen bedingt sind, so folgt daraus, daß die Körperhöhe ein polymer bedingtes Merkmal ist. Hierin unterscheidet sich die Körperhöhe nicht von den meisten normalen Eigenschaften, die ebenfalls polymer bedingt zu sein pflegen.

In zweifacher Hinsicht weichen jedoch die empirischen Verteilungskurven der Körperhöhe von der theoretischen Normalverteilung etwas ab, und zwar zeigen sie

1. einen Exzeß, das heißt, die mittleren Werte und die Extremwerte nach oben und nach unten sind häufiger, als bei Normalverteilung zu erwarten wäre,
2. eine Asymmetrie zugunsten der extrem kleinen Werte.

Diese Abweichungen sind quantitativ nicht sehr bedeutsam, aber doch auffallend und regelmäßig genug, um einen Deutungsversuch zu rechtfertigen, zumal die bisher gegebenen Deutungen nicht recht befriedigen können.

Gini hat an sehr großem Material aus Italien, Frankreich, der Schweiz, Österreich und der Türkei den Exzeß („Hypernormalität") der Verteilungskurven der Körperhöhe nachgewiesen. Er erörtert verschiedene Möglichkeiten, wie das Zusammenwirken von Erbfaktoren

und Umweltfaktoren einen Exzeß der Verteilungskurven bedingen könne, ohne jedoch zu einer plausiblen Lösung zu gelangen. Mir scheint, seine Diskussion des Problems kann in einem Punkt noch ergänzt werden.

Die Form der Verteilungskurve eines Merkmals ist nicht nur von der Häufigkeit der das Merkmal bedingenden Faktoren erblicher und peristatischer Natur abhängig, sondern auch von der Kombination dieser Faktoren untereinander. Nur bei rein zufälliger Verteilung wäre eine Normalkurve zu erwarten. Wenn aus irgendwelchen Gründen die Faktoren, die unterdurchschnittliche Körperhöhe bedingen, häufiger miteinander kombiniert auftreten würden, als bei zufälliger Kombination zu erwarten wäre, und wenn das gleiche für die Faktoren gelten würde, die überdurchschnittliche Körperhöhe bedingen, so wäre ein Exzeß der Verteilungskurve zu erwarten. Eine solche überzufällig häufige Kombination gleichwirkender Faktoren ist aber für die Körperhöhe des Menschen nachgewiesen. Es besteht eine deutliche Homogamie bezüglich der Körperhöhe, das heißt, kleine Menschen neigen dazu, ebenfalls kleine zu heiraten, große Menschen dazu, ebenfalls große zu heiraten. Die Homogamie bezüglich der Körperhöhe wurde erstmals von *Pearson* in England nachgewiesen. Später wurden die Untersuchungen von *Genna*, *Tomici* und *Uggè* in Italien, von *Schwidetzky*, *Tettenborn*, *Scheidt* und *Trin* in Deutschland, von *Willoughby* in den Vereinigten Staaten, von *Nicolaeff* in Rußland und von *Fantham* und *Porter* in Südafrika und Canada bestätigt. *Gini* fand nur eine geringe Steigerung der Häufigkeit der extrem Kleinen und extrem Großen gegenüber der bei Normalverteilung zu erwartenden Häufigkeit. Dazu ist zu sagen, daß seine theoretischen Normalkurven unter Zuhilfenahme der empirischen Mittelwerte und der empirischen Streuung konstruiert wurden, die empirische Streuung ist aber bereits infolge der Homogamie größer als die unbekannte Streuung, die man bei völliger Durchmischung der Bevölkerung („Panmixie") finden würde. Die theoretischen Normalverteilungskurven von *Gini* sind also nicht die Normalverteilungskurven, die man bei Panmixie erwarten würde, sondern sie weichen von diesen in systematischer Weise ab. Hieraus folgt aber, daß die durch die Homogamie bedingte Abweichung der Verteilungskurve von der bei Panmixie zu erwartenden größer ist als die Abweichung der empirischen Verteilungskurven von den „theoretischen" von *Gini*.

Die Homogamie kann zwei prinzipiell verschiedene, methodisch aber kaum zu trennende Ursachen haben. Einmal handelt es sich um eine mehr oder weniger bewußte Berücksichtigung der Körperhöhe

des Partners bei der Ehewahl („assortative mating"), zum andern aber um eine mittelbare Folge der Tatsache, daß Heiraten meist in geographisch, sozial oder beruflich engen Gruppen erfolgen, die unter sich in der Körperhöhe verhältnismäßig ähnlich sind. Obwohl die angeführten Untersuchungen eine solche Trennung der Homogamie nach ihren Ursachen nicht gestatten, erscheint es richtig, sie begrifflich zu vollziehen.

Dahlberg hat das Problem der „Isolate", das heißt der untereinander relativ ähnlichen Teilpopulationen, innerhalb derer die Ehen zu erfolgen pflegen, ausführlich analysiert und dabei besonders auf einige Konsequenzen hingewiesen, die sich aus der zunehmenden Auflösung der Isolate in den letzten Generationen ergeben. Eine weitere Konsequenz kann man in der Abnahme des Exzesses der Verteilungskurven der Körperhöhe sehen. Für die italienischen Rekruten des Geburtsjahrganges 1854 betrug nach *Gini* der Exzeß der Körperhöhenverteilung 3,350, für die des Jahrgangs 1920 aber nur noch 3,231. Ein Exzeß von 3,1416 ($= \pi$) würde dabei eine Normalverteilung bedeuten. Interessant ist auch, daß in einsamen ländlichen und Berggebieten Italiens, in denen die Auflösung der Isolate noch nicht so weit fortgeschritten sein dürfte, der Exzeß der Verteilungskurve der Körperhöhe am höchsten war, während er in der gemischteren Bevölkerung der Städte am geringsten war.

Auch die Asymmetrie der Verteilungskurven der Körperhöhe könnte mit der Homogamie zusammenhängen. Wir wissen, daß es eine ganze Anzahl verschiedener rezessiver Anlagen zu Kleinwuchs gibt. Diese Anlagen kommen also nur bei Homozygotie zur Manifestation. Die Häufigkeit der Homozygotie steigt aber mit dem Grade der Homogamie, sie sinkt mit der Auflösung der Isolate. Als Folge der zunehmenden Durchmischung der Bevölkerung wäre daher eine Abnahme des Kleinwuchses zu erwarten. Das ist aber genau das, was *Costanzo* in seinen umfangreichen Untersuchungen über die Körperhöhenverteilung der italienischen Rekruten vom Geburtsjahrgang 1854 bis 1916 gefunden hat. In diesem Zeitraum ist die Asymmetrie der Verteilungskurven, die fast ausschließlich auf einem Überwiegen der extrem Kleinen beruht, weitgehend zurückgegangen. Ein weiteres wichtiges Ergebnis von *Costanzos* Arbeit ist die Abnahme der Streuung der Körperhöhe in den letzten Dezennien, auch dieser Befund ließe sich durch die zunehmende Durchmischung erklären.

Dahlberg sieht auch in der Körperhöhensteigerung in den letzten 100 Jahren im wesentlichen eine Folge der Auflösung der Isolate.

Zusammenfassung.

Die Verteilungskurven der menschlichen Körperhöhe weichen in charakteristischer Weise von einer Normalverteilung ab. Diese Abweichungen lassen sich dadurch erklären, daß in menschlichen Bevölkerungen bezüglich der Körperhöhe keine Panmixie sondern eine relative Homogamie herrscht. Mit dieser hypothetischen Erklärung stimmt die Tatsache überein, daß mit zunehmender Durchmischung der Bevölkerung in den letzten Generationen die Verteilungskurven der Körperhöhe dem Normaltyp nähergekommen sind.

Resumé.

Les courbes de la distribution de la stature humaine s'écartent de la distribution binomiale d'une manière caractéristique. Les déviations peuvent être expliquées par le fait qu'il n'y a pas de panmixie absolue dans des populations humaines à l'égard de la stature mais une homogamie relative. Que le mélange augmenté des populations pendant les générations dernières a rapproché les courbes pour la distribution de la stature humaine de celle de la distribution binomiale s'accorde avec cet explication hypothétique.

Summary.

The curves of the distribution of human stature diverge slightly from the binomial curve in a characteristic manner. These divergences may be explained through the fact that for stature panmixia does not quite obtain in human populations but a relative homogamy. With this hypothetic explanation agrees the fact that through increased mixing of the population during the last generations the curves of the distribution of human stature seem to have approached the binomial curve.

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BLOOD COAGULATION IN CONDUCTORS OF HAEMOPHILIA

By ERIK SKÖLD

In his large monograph on haemophilia, *Schlossmann* [1930] surmised that conductors had a prolonged coagulation time. It was said to be normal in only 2 amongst 13 conductors. Later authors who have studied the matter, and their results, are listed in table 1.

Table 1. Conductors with Prolonged Coagulation Time.

Author	Number of examined conductors	Prolonged coagulation time in
Traum, Schaaf and *Linden* [1931]	4	4
Studt [1937]	16	12
Løfgren [1937]	2	2
Günder [1938]	14	9
Andreassen [1943]	31	30

In evaluating data such as these one must first take into account the method for evaluating the coagulation time which was used by the investigator concerned. Unfortunately none of the methods available are particularly exact. The principle of the thing is to determine and record the time it takes for a drop of blood to clot under a variety of conditions. The normal values used for purposes of comparison are, of course, themselves none too reliable; and actually the various figures given are merely different limits of normal variation. This partly is so because the authors have used slightly dissimilar methods. Apart from those I published in 1944, no normal figures based on statistical analyses seem to exist. My figures were obtained with the so-called Hedenius's method. The mean for normal men was 5 ½ minutes (5.47) with a standard deviation of 1 ½ minutes (1.47), and for normal women the mean was 5 minutes (4.91) and the standard deviation just over 1 ½ minutes (1.70). Though not significantly different, the

mean for the conductors in my series was probably different from the normal mean (difference 1.06 $\pm$ 0.35). At least in my material and with the method used, it evidently was possible to diagnose only some of the conductors.

Andreassen gave no figures for a theoretically normal distribution, but his diagram supports the view that the coagulation time in normal women may be as long as 8 minutes. He also gave figures for 31 established conductors, a good number of whom showed values that were close to or on the verge of being normal, but over half the cases seem to deviate markedly from the normal.

Since—as demonstrated by *Dahlberg* [1949]—a number of women who were thought to be conductors merely are mothers of mutations, it may be justified to take up the subject for review.

In order to make the reader realize the nature of the method and how inaccurate it may be, I have had one of my assistants out making coagulation time determinations. Because clotting is a gradual process and not an instantaneous transformation, the method will at best suffer from quite a high order of inaccuracy.

My series comprised those haemophiliacs and their relatives who are known to the Swedish blood banks (or blood collecting units) of which there currently are 18. Owing to blood transfusions' vital importance to haemophiliacs, it may be assumed that nowadays all of them keep in touch with the blood banks and that all cases existing in the country therefore are known. Haemophiliacs are particularly often troubled by intraarticular haemorrhages that wear down the joints and immobilize the patient. All that is needed to stop this condition is a simple blood transfusion. Other types of haemorrhage obviously lend themselves to the same treatment, not to speak of tooth extractions and other operations which would be out of the question unless preceded by blood transfusions.

In distinguishing between conductors and normal women one must, as previously noted, have a set of normal figures as a standard of reference. For this purpose I determined the blood coagulation time for 101 women donors among the clientèle of the St. Erik blood bank in Stockholm (Bergquist's method). The mean proved to be 11.2 minutes and the standard deviation 1.5 minutes. The normal coagulation time is in other words practically 11 minutes with a variation of 4.5 minutes: thus, up to 16 minutes and down to 7 minutes.

It turns out that the figures for established conductors differ from

the figures for supposed conductors, i.e. women with haemophilic sons. The mean for conductors was 13.2 and the standard deviation 2.35. The difference between the means is 2 ± 0.46 and statistically significant. Separately, however, the conductors showed figures of which 75 per cent lay within normal limits, i.e. below 16 minutes. Such a transgressive variation must in part mean that some of the women we had regarded as conductors actually were mothers of mutations. Another possibility is that the difference between true conductors and normals is far too small to warrant any conclusions with regard to whether or not a separate individual is a conductor.

The preceding data were announced at the Eighth International Congress of Genetics at Stockholm, 1948, and having now checked up on my facts I have found the following.

The standard error of measurement proved now to be 1.31 minutes, i.e. 9.2 per cent on a mean of 14.30 ± 0.36 minutes for men or 9.5 per cent on a mean of 13.76 ± 0.92 minutes for women. In normal women the range of variation can be put at 4.81 minutes and in normal men at 3.45 minutes. For conductors the mean is 19.73 and the variation characterized by a σ of 7.13. Thus from both normal men and women the difference is significant. The three series of normal values given above (1944, 1948 and 1952) are a little different because of different size of the materials and random variations and also because of slightly different methods used. The most interesting point is how many of the conductors that fall outside the range of normal variation for the two sexes. This is shown in table 2 which reveals how many fall outside the limits of 2 and 3 σ respectively. Compared to the normal values for men 35.3 per cent fall outside 2 σ and

Table 2. Number of conductors with coagulation times outside the range of variation up to $M \pm 2\sigma$ and $M \pm 3\sigma$ (calculated on the mean and standard deviation for men as well as on those for women).

Conductors compared with the mean for	Range of variation	Number of determinations (= N)	1st value of which outside the range of variation	
			Number	% of N
Men	$M \pm 2\sigma$	34	12	35.3
	$M \pm 3\sigma$	34	6	17.6
Women	$M \pm 2\sigma$	34	9	26.5
	$M \pm 3\sigma$	34	5	14.7

17.6 per cent outside 3 σ, and compared to the normal values for women 26.5 per cent fall outside 2 σ and 14.7 per cent outside 3 σ. These discrepancies are due to the differences between the normal values for men and women. The small deviations from the figures given earlier in the investigation are probably due to random variation. This point is clearly shown in the diagram of the results obtained for 34 conductors and the normal values for men. It discloses that the coagulation times for the majority of the conductors fell within the range of normal variation.

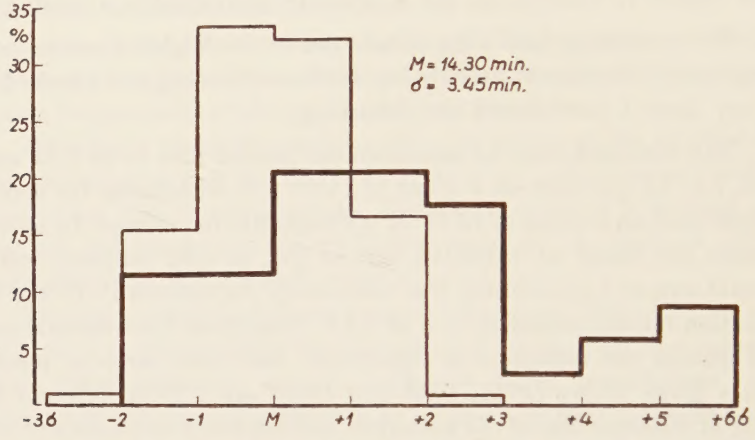

Fig. 1. Normal blood coagulation times for men (thin line) and for established conductors (thick line), percentually distributed according to different range of variation limits from the mean of the normal figures.

The question as to conductors' coagulation times is of considerable practical importance, since such women nowadays are sterilized in some countries. Such being the case it is justified to warn against taking too much notice of moderate deviations from the normal. In the separate case I for one would hesitate to return a definite judgment without repeated tests. A correct assessment of the situation would almost always be dependent upon an estimate of the individual variability from one occasion to another. Let it be remembered that this individual variability seems to be very considerable in haemophiliacs.

Summary.

The mean of the coagulation times for hemophilic conductors is significantly different from the mean for normal persons which has been pointed out previously although without giving exact figures.

The present investigation shows that in separate cases one single determination is far from always enough for deciding if women are conductors or not and that consequently one cannot depend on single determinations for sterilization. This agrees with *Dahlberg*'s investigation (1948), in which he pointed out that most of the hemophiliacs are due to mutation.

Résumé.

Une différence significative peut être constatée entre le temps moyen de coagulation du sang chez les femmes susceptibles à transmettre la hémophilie (les conductrices de la maladie) et le temps moyen de coagulation chez des personnes normaux, ce qui a été signalé auparavant mais sans qu'on ait donné des chiffres exacts. L'étude présente montre que dans un cas particulier une seule détermination n'est pas toujours assez pour décider si une femme est conductrice de la maladie, et on ne peut pas donc faire stériliser des femmes sur la base de déterminations seules. Cela est conforme à l'étude de *Dahlberg* [1948], dans laquelle il souligne que la plupart des hémophiles sont des mutations.

Zusammenfassung.

Der Mittelwert der Blutgerinnungszeiten für die Hämophilie-Konduktoren unterscheidet sich deutlich von dem Mittelwert für normale Personen, welches schon früher aufgezeigt wurde ohne genaue Ziffern anzugeben. Die vorliegende Untersuchung zeigt, daß in verschiedenen Fällen eine einzelne Bestimmung nicht hinreichend ist um zu entscheiden, ob Frauen Konduktoren sind oder nicht und daß man sich folglich im Falle von Sterilisation nicht auf einmalige Bestimmungen stützen kann. Dies stimmt mit der Untersuchung von *Dahlberg* [1948] überein, in welcher er hervorhebt, daß in den meisten Fällen von Hämophilie Mutation zugrunde liegt.

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THE SWEDISH JEWS

A discussion of the basic concept.

By GUNNAR DAHLBERG

Particularly some years ago, when Sweden too had its quota of Nazi sympathizers, it was often asked whether the Jews really were a race. The question frequently came from members of the so-called socialist intelligentsia who detested anti-Semitism and required an authoritative reassurance that the Jews were not a distinct race.

Obviously the true retort to anti-Semitism is that men should be judged not by their descent but on their merits. The same applies by the way when men and women are compared. It would be nice to find a shortcut to a judgment upon our fellow men—but none exists. The popular interest in constitutional types might well be based on a similar mental outlook, but constitutional types simply ask for misinterpretations.

But let us return to the matter of the Jews. The answer to our question will vary depending on our definition of "race". As a matter of fact people are prone to regard as distinct races divisions of the population that have special traditions and a culture which in any way differs from their own.

When we were discussing the race question at Paris in the foresummer of 1951 it was proposed from some quarters that the term "racial groups" should be replaced by "ethnical groups". But this met with opposition on the grounds that the ethnical groups, or cultural groups, were reasonably well defined and not in agreement with the racial groups. The argument was advanced that the lay public is apt to confuse racial groups and ethnical groups; but as scientists we were only concerned with the scientific aspects of race, and such a misconception was none of our affair. We were eleven scientists who met at Paris on the invitation of UNESCO. The majority of the delegates (seven) were anthropologists, i.e. students of race, while a smaller number were geneticists, i.e. students of heredity, who had been particularly active in the field of human genetics.

Race has long been defined thus: A race is a people or division of

humanity which differs from others in respect of hereditary characters. This definition has been proposed by, for example, *Martin*, the renowned anthropologist. Almost the same definition is used by *Fritz Kahn* in "Jüdisches Lexikon" [1930]. No conclusive proof exists that the characters one uses actually are hereditary. Stature for example is widely used as a racial character, but it has not been determined how far stature is influenced by hereditary factors alone. There are grounds for supposing that it may be influenced by environment, particularly during the formative years. In other words it is a highly dubious racial character. A number of other important racial characters suffer from similar weaknesses.

In anthropology one operates with the concept "pure races", which by assumption existed in bygone days, and "mixed races" which are said to be prevalent in our times. However, there is no proof that pure races ever existed.

Anthropology is moreover based on an obsolete notion of heredity which seldom is clearly declared. In pre-Mendelian days it was thought that the germ plasm was a substance capable of being diluted, so that half-bloods, quarter-bloods, etc. were produced. Anthropology indeed antedates Mendelism by far. According to the latter theory the germ plasm is composed of particles that cannot be diluted but merely intermixed in various ways. One of the few that lucidly have discussed the two theories is *Weinberg* [1909] who spoke of "vermischte Vererbung", viz. that which I prefer to call the substance theory, and of "alternative Vererbung" whereby he meant Mendelism. Upholders of the substance theory seem to have believed that different races had different germ plasm. Thus, according to the substance theory, each separate individual would be recognizable as a Jew. Individuals could obviously differ from each other—just as objects of the same metal may be either decorative things or kitchen utensils although basically they are of the same substance. By this early theory the Jews therefore were a mixed group historically, which down the ages became more and more intermingled.

I have long advocated another definition of race (published 1941). It is essential clearly to set out what one means by hereditary on the one hand and by a division of mankind on the other. As I see things, a race may be defined as an isolate, or a subdivision of an isolate, that differs genetically from other isolates. An isolate denotes a set of people among whom matings take place. An isolate may be geographically or socially bounded, but the boundaries need not be

sharp. A small number of matings may of course go beyond the boundaries. The isolate is in other words a fairly new conception which has been analyzed mathematically (cf. *Dahlberg* [1938]), but going into details would carry us beyond the scope of this paper. It will suffice to mention that respectively tall and short individuals in a people are not called separate races: they do not constitute distinct groups whose members predominantly mate among themselves. They are not in fact isolates.

Hereditary or genetic dissimilarities may vary with regard both to type and to degree. It may be that one or more genes for a character occur exclusively in one race and not in others. If a race is thus distinguished we speak of a race difference of the first degree. Such races are in other words not far from species. Negroes, Mongols, etc. are reckoned as races of the first degree. Equally clearcut race differences of the second degree do not exist however. The latter are dependent on the presence of one or more genes in unequal numbers in different isolate groups. It has been debated whether one genetic difference is all that is required. So far as I can see it would be incorrect to distinguish between genes of different types. Suppose, for instance, that a gene produces dwarfism, which would seem to be quite enough for a racial character. Other genes have less striking effects, but it is impossible to draw a line between genes with considerable effects and genes with less remarkable manifestations. On the whole we know little about the frequency of genes in populations. Even if the new definition is clearer than the old, the empirical basis for racial classification is too weak. Yet it would be vain on such grounds to deny the difference between Negroes and Mongols, but the genetical aspects of the difference are too imperfectly understood to enable one to form a clear picture. One can only say that differences exist without going into specific details. The notion of race is in other words a makeshift which we have adopted to give a name to obvious differences between populations.

To my mind the race concept will undoubtedly be developed in a genetic direction. It is indeed a part of genetics. Those genes whose frequency we know most about are the ones that govern different blood groups. Races are already being discussed on that basis. Based mainly on these genes, a book on races has recently been published by *Boyd* [1950] in the U.S.

Using the genetic definition of race, one cannot with regard to secondary racial characters say anything definite about individuals,

only about peoples. In, say, Sweden it is obvious that any differences that there might be between Jews and other inhabitants at most can be designated as differences of the second degree. There exist pigmentational differences and other peculiarities of appearance which certainly have genetic causes, but the frequency of the genes is not such that it seems possible in the individual case to distinguish between persons of Jewish and of Swedish descent. (Cf. below.) This means that the difference is very insignificant and almost immaterial. Besides, it is quite possible that between Jews from different countries there are differences of the same magnitude, even if as yet there is not enough to go on for a scientific analysis. It would thus be quite reasonable to suppose that Jews from Yemen differ more from, say, Swedish Jews than the latter differ from Swedes. The Jews are as a matter of fact by some considered as a racial group, i.e. they make up several races of the second degree whose specific differences are rather insignificant. Whether this actually is so has not been investigated. But I have tried to arrange such a study on the Yemenites that of late have immigrated to and settled in Palestine.

It has long been alleged that Jews have a peculiar propensity for diabetes. Recent researches have nevertheless shown that such is not the case (*Bieneck* [1940]). The high incidence of diabetes amongst Jews is solely due to the fact that they oftener seek medical advice, which in turn probably is because the set of Jews studied happened to be fairly well off. Diabetes was therefore detected oftener in them, but not oftener than in the upper levels of society as a whole. If the Jews instead are compared with racial groups on the same cultural and economic levels, no difference will be found with regard to the incidence of diabetes. This incidence is actually correlated very closely indeed to the social status (cf. *Dahlberg* [1950]). There is no reason to suspect that the differences in diet should cause an unequal frequency of diabetes.

The bond that has kept the Jews together is obviously their religion; and as a result of that and of anti-Semitism the Jews have a different sociological tradition and have been subjected to particularly strict selection, which may have created genetic differences. The traditional characters are of course immaterial racially. Whether selection gives rise to differences is not known. It is possible, however, that selection tends to destroy the slow-witted and that consequently intelligence is more common among Jews. Yet in the few investigations made intelligence tests have disclosed no essential differences

between different races. In no. 3 of vol. 9 [1951] of the American Journal of Physical Anthropology *H. H. Strandskov* and *S. L. Washburn* say that "there may appear to be a conflict between the methods of genetics and anthropology. But this appearance is superficial and misleading." I think that this statement is over-optimistic.

Let me round this off with a few words about race and patriotism. When talking about their own nation persons are liable to claim that the people is gifted with some character or peculiarity that is valuable to mankind. It is not stated flatly that this gift is hereditary, but it is easy to surmise that this is what is meant even if it is not put into so many words. One feature of patriotism is liable to be a firm belief in the great worth of the race to which belongs the person concerned, although this is not always the case. One may for example echo the American and talk about the greatness of one's "way of life" by which (presumably) he refers to a composite of mental outlook and tradition. In a manner of speaking the vague phraseology of those who trade in patriotism brings to the fore the notion of race, so that people who take not the least bit of interest in racial problems have them drummed into their consciousness. Perhaps it may be said that patriotism keeps alive the racial concept in a roundabout and wholly unjustified manner.

The Swedish Jews from an Anthropometric Viewpoint.

As can be seen from the above it is surprising that the Jews are not more investigated from an anthropometric viewpoint considering the role they have played in cultural life, and with regard to anti-semitism. There are, of course, an unlimited number of more or less fantastic statements about them, but very few real investigations. As regards the Swedish Jews, there has been no anthropometric investigation. The older anthropologic literature has been reviewed by *Fishberg* [1913]. The later literature is given by *Ruppin* [1931]. In *Eickstedt* [1934], for example, there are no numerical statements. He considers the Jews as members of the Oriental race. *Coon* and others state that the Jews are a subdivision of the Mediterranean race.

In general when one speaks about the "Semitic" race the Jews who lived in the near Orient are meant, but this usage is not clear. The Jews who have immigrated to Europe have come here by two separate ways; some came over Poland and Russia (the so-called Ashkenazic Jews) and others over Spain (the so-called Sephardic Jews). The Swedish Jews seem to belong to the former group. It

should also be pointed out that the Jews in Palestine have passed through a much changing history and have been mixed with other peoples to a high degree. By mixing with Amorites (according to *Haddon* [1924]) they are assumed to have got a certain percentage of light-haired and light-eyed individuals. However, this is only a hypothesis advanced by *von Luschan* [1892]. As a matter of fact we don't know if the Amorites really were blond. Anyhow, the old Jews arose through a mixture of different peoples and they have of course become more and more mixed ever since.

In the years 1922–1924 I was employed at the State Institute for Human Genetics and Race Biology. My main task at this time was the organization of a collection of anthropometric material from draftees in the Swedish army. I measured about half the material myself. *Lundborg-Linders'* "The Racial Characters of the Swedish Nation" [1926], mainly written by *S. Wahlund*, was founded on this material.

I have sorted out the Jews included in this investigation by choosing those of Jewish religion (which was noted on their cards) and by choosing those with Jewish names. In examining this material I

Table 1. Distribution of pigmentation in Jews, Probable Jews and Swedes.

Degree of pigmentation	Jews		Probable Jews		Swedes	
	Number	%	Number	%	Number	%
			Colour of the iris			
1	7	22.6±7.5	6	40.0±12.7	41437	86.9±0.16
2	15	48.4±9.0	4	26.7±11.4	3861	8.1±0.13
3	9	29.0±8.2	5	33.3±12.2	2364	5.0±0.10
Total	31	100	15	100	47362	100
			Head hair colour			
Light	5	16.1±6.6	3	20.0±10.3	34126	72.7±0.21
Dark	26	83.9±6.6	12	80.0±10.3	12828	27.3±0.21
Total	31	100	15	100	46954	100
			Eyebrow colour			
Light	6	19.4±7.1	2	13.3±8.8	34011	72.4±0.21
Dark	25	80.6±7.1	13	86.7±8.8	12956	27.6±0.21
Total	31	100	15	100	46967	100
			Pubic hair colour			
Light	5	16.1±6.6	3	20.0±10.3	33239	70.8±0.21
Dark	26	83.9±6.6	12	80.0±10.3	13714	29.2±0.21
Total	31	100	15	100	46953	100

have had assistance from Professor *Hugo Valentin* and the genealogist Miss *Ella Heckscher*, both of whom have a very thorough knowledge of Swedish Jews. Those who were selected were divided into two groups, Certain Jews (all born in Sweden) and Probable Jews, and analyzed. The result of this analysis can be seen from the following tables.

As has been pointed out in earlier works one can distinguish between two types of inherited characteristics, namely 1) those that influence the pigmentation of the cells or the quality of the cells in some manner, and 2) those that influence cell proliferation during development and give differences in size and shape of the individuals.

The present material has the advantage of having been collected by trained personnel with no preconceived view regarding the purpose of the investigation since anti-semitism was not a current issue in Sweden at that time.

We will begin by considering the pigmentation of the iris. It can be seen from table 1 that the group Certain Jews as well as Probable Jews have a smaller per cent of individuals belonging to group 1, i.e. those possessing blue or grey eyes. The difference is significant but is somewhat less pronounced for Probable Jews. Those belonging to group 2, i.e. those having mixed-colour eyes, are over-represented in Certain Jews as well as Probable Jews. The same is the case with those belonging to group 3, the brown-eyed group. Also here the difference runs parallel, that is to say Certain Jews as well as Probable Jews have a higher per cent of brown-eyed individuals than other Swedes.

A similar situation is found as regards hair colour. The relatively light-haired individuals are more numerous on a percentage basis among the Swedes than among Probable Jews, while on the other hand dark-haired individuals are more numerous on a percentage basis among Certain Jews than Probable Jews. This is true for head hair as well as eyebrows and pubic hair. Despite the fact that the material is small, the differences are statistically significant.

It should be pointed out that the measure figures are not always given in *Lundborg-Linders* [1926]. The height of the symphysis has been used in calculating leg length by adding 3 ½ cm. Body trunk length has been calculated by subtracting symphysic height from suprasternal height, and arm length by subtracting dactylon height from acromium height. The actual measured figures are given here for Jews more for the sake of completeness, but the measurements

Table 2. Measurements of the body-build of Swedish Jews, Probable Jews and Swedes.

Measurements of the body-build	Jews		Probable Jews		Swedes	
	Mean ± standard error	Standard deviation	Mean ± standard error	Standard deviation	Mean ± standard error	Standard deviation
Stature, mm	1697.6±13.3	73.9	1671.7±18.3	70.8	1722.2±0.3	59.4±0.2
Suprasternal height, mm	1385.3±11.4	63.5	1368.4±17.5	67.6	1408.9	.
Height of symphysion, mm	858.4±8.8	48.8	859.3±12.2	47.1	885.2±0.2	43.0±0.1
Height of acromion, mm	1367.3±11.7	65.1	1360.0±14.5	56.3	.	.
Height of dactylion, mm	609.5±7.3	40.3	604.4±8.0	31.0	.	.
Bi-acromial diameter, mm	392.2±3.0	16.5	385.9±4.1	15.8	392.3±0.1	16.7±0.1
Inter-iliocristal breadth, mm	279.4±2.6	14.6	280.4±3.5	13.6	288.0±0.1	15.2±0.0
Trunk length, mm	526.9±4.9	27.5	509.1±9.2	35.6	523.7±0.1	24.1±0.1
Arm length, mm	756.8±5.8	32.5	755.6±10.4	40.4	784.6±0.2	33.4±0.1
Leg length, mm	893.4±8.8	48.8	894.3±12.1	47.0	920.2±0.2	43.0±0.1
Span, mm	1905.8	.	1897.1	.	1961.5	.
Bi-acromial diameter index	23.09±0.16	0.90	23.10±0.16	0.63	22.80	.
Trunk length index	31.01±0.22	1.25	30.44±0.38	1.46	30.49±0.01	1.18±0.00
Arm length index	44.65±0.18	1.01	45.20±0.37	1.43	45.54	.
Leg length index	52.61±0.22	1.25	53.49±0.39	1.52	53.43	.

mentioned above have also been calculated to make comparisons with the entire Swedish material possible. In viewing the body measurements that have been taken (see table 2), one finds that the stature is less in Certain Jews and Probable Jews than in Swedes. For the combined material of the two Jewish groups, a significant difference of 33.0 ± 10.9 mm is found. Since the Jews are included under the material of the Swedes, the Jews fall within the range of variation of the Swedes, but still the differences between the average values are significant. A diagram of the per cent distribution in Jews as well as Swedes (fig. 1 and 2) is also given and from this it is seen that the Jews in general fall among the lower measurements. Such differences, more or less pronounced, can be found in most body measurements. This is, of course, natural since they are related to body size. One measurement which shows no difference is the per cent leg length (leg length index). On the other hand, arm length and arm length index show that the Swedish Jews have slightly shorter arms than the rest of the Swedish population. The Jews also appear to have slightly narrower shoulders and a slightly smaller pelvic width (the last difference being statistically significant) when compared to other Swedes. The difference, however, is not larger than that corresponding to the lesser stature of the Jews.

Proceeding on to the head measurements (table 3), we can see that the head length of Jews is 3.9 ± 1.1 mm shorter, while the head

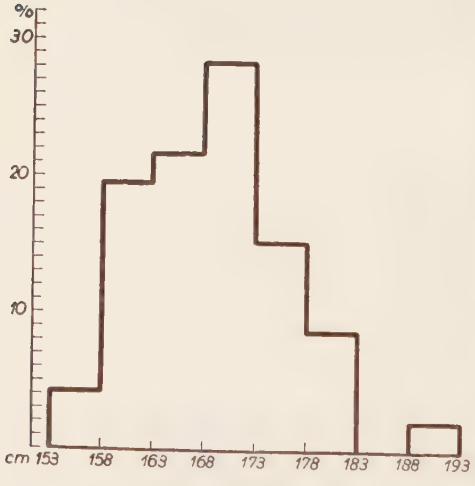

Fig. 1. Percentual distribution of stature in Swedish Jews.

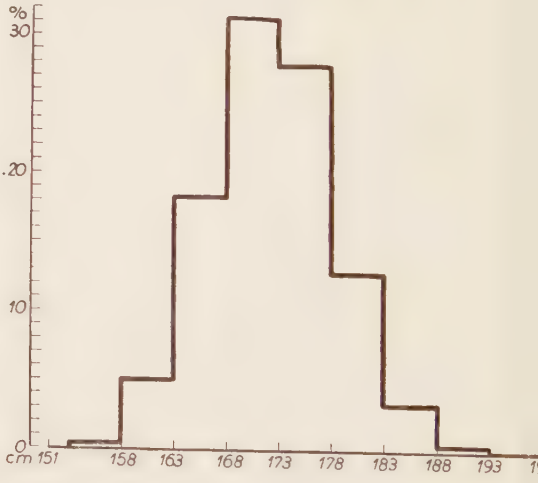

Fig. 2. Percentual distribution of stature in Swedes (the whole population).

Table 3. Measurements of the head of Swedish Jews, Probable Jews and Swedes.

Measurements of the head	Jews		Probable Jews		Swedes	
	Mean ± standard error	Standard deviation	Mean ± standard error	Standard deviation	Mean ± standard error	Standard deviation
d length	190.0±1.4	7.9	189.6±1.7	6.6	193.8±0.03	6.2±0.02
d breadth	152.5±0.9	5.1	151.2±1.0	3.8	150.4±0.02	5.1±0.02
halic index	80.3±0.6	3.2	79.8±0.6	2.4	77.7±0.01	3.1±0.01
imum frontal diameter .	103.2±0.7	3.7	104.3±1.3	4.9	104.6±0.02	4.3±0.01
e breadth	136.2±0.9	4.7	135.6±1.1	4.2	136.0±0.02	4.8±0.02
phological face height .	124.4±1.3	7.3	124.7±1.6	6.0	126.6±0.03	6.9±0.02
phological face index . .	91.3±1.1	5.9	92.0±1.4	5.4	93.1±0.03	5.6±0.02
onial diameter	104.2±0.7	3.7	104.9±1.5	5.7	.	.
o-frontal index	75.8±0.5	2.7	76.9±0.9	3.5	77.0±0.04	2.9±0.03
o-mandibular index. . .	76.7±0.5	2.7	77.4±1.2	4.7	76.0±0.04	3.4±0.03

width is somewhat greater, the difference not being significant. Thus it may appear that the Jews have a slightly larger head index (the difference is 2.4±0.4%, thus being significant although small). However, the head measurements in general do not show any significant differences.

In 1910 there were 3,484 confessors of the Mosaic faith in Sweden and in 1930 the number was 3,396 according to official statistics. In addition, there are in Sweden an unknown number of descendants of those of the Mosaic faith who have now joined the Swedish Christian State Church, and furthermore a number of immigrants who are not Swedish citizens. While an exact calculation cannot be made, the figures quoted indicate that most of the Jewish conscripts have been included in our investigation.

Summary.

In summary it should be stressed that the differences that have been demonstrated between Certain Jews, Probable Jews and Swedes are not of any great significance. On an average the Jews are more dark-eyed and dark-haired and have a smaller stature, but the differences are not of such a kind that one can in separate cases distinguish between Jews and Swedes on this basis. It is obvious that Jews who have come to Sweden have undergone considerable mixing in times past. Since this is true of their physical characteristics one has every reason to believe that it holds true as regards possible psychic differences. Even if the Jews in olden times belonged to a special race

(the Oriental or Mediterranean race) there is consequently no reason to assume this for Swedish Jews nowadays.

Résumé.

Il est souligné que les différences anthropologiques trouvées entre des personnes sûrement juives, des personnes probablement juives et des Suédois ne sont pas d'une grande importance. En moyenne les juifs ont les yeux et les cheveux plus foncés et sont d'une taille plus petite, mais les différences ne sont pas de nature telle qu'il soit possible de distinguer un juif d'un Suédois dans chaque cas particulier. Il est évident que les juifs venus en Suède ont été considerablement mélangés dans le passé. Cela étant valide pour les qualités physiques on a lieu de croire qu'il est aussi applicable quand il s'agit de différences psychiques possibles. Même si les juifs appartenaient dans les anciens temps à une race particulière (la race orientale ou méditerranéenne) il n'y a donc aucune raison de supposer que cela soit valable pour les juifs suédois d'aujourd'hui.

Zusammenfassung.

Zusammenfassend soll hervorgehoben werden, daß die Unterschiede, wie sie zwischen „Gewiß-Juden", „Wahrscheinlich-Juden" und Schweden aufgezeigt werden, nicht von großer Bedeutung sind. Im Durchschnitt sind die Juden dunkeläugiger und von dunklerer Haarfarbe und besitzen eine geringere Körperlänge, doch sind die Unterschiede nicht dergestalt, daß man auf dieser Basis in jedem Falle zwischen Juden und Schweden unterscheiden könnte. Es ist augenscheinlich, daß die nach Schweden gekommenen Juden im Laufe der Zeit eine beträchtliche Vermischung erfahren haben. Da dies für ihre physischen Merkmale stimmt, hat man alle Ursache zu der Annahme, daß dies auch in bezug auf mögliche psychische Unterschiede seine Gültigkeit besitzt. Selbst wenn die Juden in früheren Zeiten einer besonderen Rasse angehörten (der orientalischen oder mediterranischen Rasse), liegt doch folglich kein Grund vor, dies auch für die heutigen schwedischen Juden anzunehmen.

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From the State Institute of Human Genetics and Race Biology, Uppsala, Sweden

GENETIC INVESTIGATIONS IN DIFFERENT POPULATIONS

By GUNNAR DAHLBERG

As early as 1939 I proposed a classification of characters which I discussed at length in my monograph of 1947. The characters were divided into three groups, namely:

1. Genetic traits which are due to the presence of certain genes and unaffected by environmental factors,
2. Environmental traits which are due to the presence or absence of certain environmental factors, and
3. Constellational traits which are due both to environment and to heredity.

Fig. 1 illustrates that populational properties influence the class to which a character belongs. As an example consider paralytic dementia. It is a constellational character which probably is dependent both on the presence of certain genes that cause the particular response and on the infection with syphilis. In a population composed entirely of syphilitics the disease would be due solely to those genes

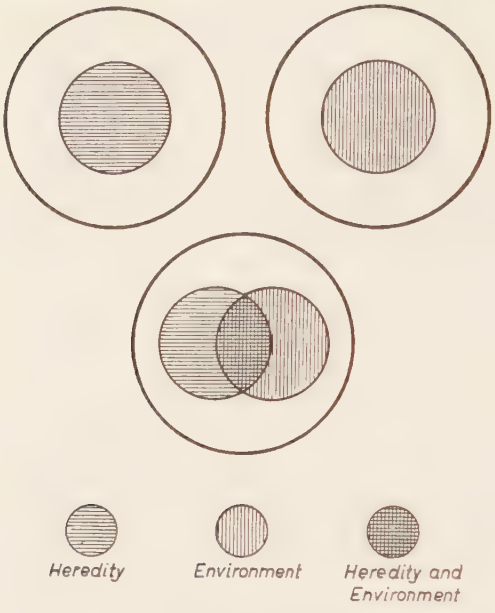

Fig. 1. Traits of different kinds. Above, to the right: a hereditary trait; to the left: an environmental trait. Below: a constellational trait.

which are responsible for the hypothetical hypersensitivity and so become a genetic character.

In my opinion this classification of characters is still adequate. It implies that one must take the observed persons' population into account.

A genetic investigation theoretically can have two aims: 1. On the basis of a more or less complete knowledge of the traits of the parents and their relatives, to forecast more or less definitely the traits which may be inherited by the child of a projected marriage; and 2. on the basis of known personal characters, to estimate the future properties of a population within which mating takes place at random more or less modified by different degrees of inbreeding, assortative mating and so on (cf. *Dahlberg* 1947).

Though studies of the latter type obviously are more important for human genetics, they have not been very numerous. I hope they will draw more attention in the future.

But the first type of problem—examination of relatives of probands with certain traits—also demands recognition of populational properties. Take for example a number of trait-carriers among whose relatives we wish to know the frequency of the trait carried. Obviously the trait will show a higher incidence in those families than in families

picked at random from the population. For in the former case we are dealing with a selected material, with persons who have received the gene from common ancestors. Similar reasoning immediately tells us that the same frequency of the trait should not be expected in relatives with different degrees of consanguinity; the frequency must be higher in close relatives than in more distant ones. However, the over-representation of the trait in the family circle depends also on the mode of inheritance (recessive or dominant) and finally on the trait's overall frequency in the population.

Of the frequencies concerned—which, by the way, are perfectly simple to compute—those are most important which refer to the relatives who are closest and therefore most easily available for examination. In the below we shall first derive the mathematical formulae for calculating the hereditary properties of the trait-carriers' ancestors and direct offspring. With regard to more distant relatives we must here be content with the properties of sibs, parental sibs and first cousins of trait-carriers.

Parents and Grandparents.

There are various ways of determining the hereditary equipment of the grandparents of certain types of zygotes. To find the ancestral proportion of genes in the different generations, we may avail ourselves of the following simple method.

Let us first suppose that the probands are homozygotes and consequently have received two similar genes, say R-genes, carrying the transmitted trait (Fig. 2). Each parental couple has four genes, two of which are R-genes. What the other two genes carry is un-

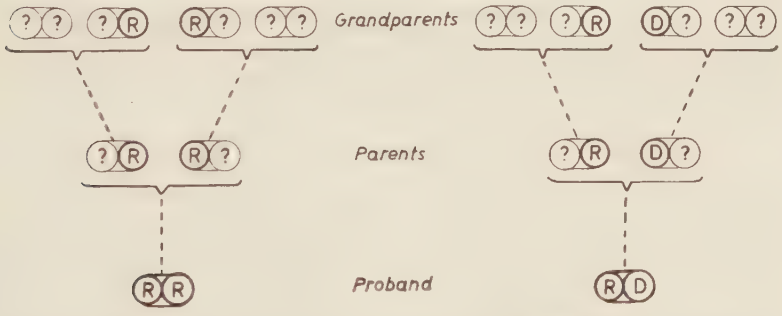

Fig. 2. Genotypical character of the ancestors (and offspring) of homozygotic, respectively heterozygotic probands. The genes indicated by ? have on the average the same character as in the population.

known, but the chances that they are R-genes must be the same as for all other genes in the population that our probands come from. If, as before, we call the relative frequency of the R-gene in the population r, we obtain the following expression for the gene frequency in the first ancestral generation:

$$r_{p_1} = \frac{2}{4} + \frac{2}{4} \cdot r = \frac{1+r}{2}$$

Similarly, among the eight genes carrying the trait in the four grandparents and among the sixteen corresponding genes in the great grandparents, two must be R-genes, i.e. those from which the proband's genes are directly inherited. The other genes, however, vary between R and D just as in the population. The relative number of R-genes must consequently be the following:

Grandparents: $$r_{p_2} = \frac{2}{8} + \frac{6}{8} \cdot r$$

Great-grandparents: $$r_{p_3} = \frac{2}{16} + \frac{14}{16} \cdot r.$$

The simple formulae for the first three ancestral generations to recessive homozygotes, as well as the general formula for the n'th generation, are given in table 1 (group a).

If the probands are dominant homozygotes (DD), we must evidently obtain formulae that are completely similar in form, except that d or $1 - r$ always must be written in place of r. For example, the

Table 1.

Probands	Gene frequency among the ancestors and children: 1st generation (parents or children)	2nd generation (grandparents or grandchildren)	3rd generation (great-grandparents or great-grandchildren)	n : th generation
1.	2.	3.	4.	5.
A. RR-zygotes	$\frac{1+r}{2}$	$\frac{1+3r}{4}$	$\frac{1+7r}{8}$	$\frac{1+(2^n-1)r}{2^n}$
B. RD-zygotes	$\frac{1+2r}{4}$	$\frac{1+6r}{8}$	$\frac{1+14r}{16}$	$\frac{1+(2^{n+1}-2)r}{2^{n+1}}$
C. DD-zygotes	$\frac{r}{2}$	$\frac{3r}{4}$	$\frac{7r}{8}$	$\frac{(2^n-1)r}{2^n}$

frequency of D-genes in parents of DD-zygotes can be had from the equation:

$$1 - r_{p_1} = \frac{2}{4} + \frac{2}{4}(1 - r) \cdot \cdot \cdot r_{p_1} = \frac{r}{2} \text{ (Cf. group C in table 1).}$$

Formulae for ancestors of heterozygotes (RD) may be derived in a similar manner (Fig. 2). Here too the nature of two genes in each generation is known, although one is an R-gene while the other must be a D-gene. With regard to the remaining genes we must expect the same proportions between R-genes and D-genes as in the population at large. Thus in the parents the relative number of R-genes is $\frac{1}{4} + \frac{2}{4}r$, and in the grandparents $\frac{1}{8} + \frac{6}{8}r$, etc. (cf. group B in table 1).

Briefly to sum up now the purport of the given formulae and of fig. 2, we may say: "If we trace backwards the genes in an individual or in a group of individuals with a similar hereditary background, new genes from the population will be added for each generation so that the number of genes is continually being doubled. And thus the proportions of R-genes and D-genes are getting ever closer to the proportions in the population."

Since we now would seem to have studied the gene frequency in parents, grandparents, etc. of a particular type of zygotes, it might seem that by inserting the figures in the general formulae given above we could compute the zygotic distribution of the respective ancestors and their children. Such is not the case, however, for the formulae provided in the foregoing presuppose that the examined subjects are in no way selected, whereas the persons to be studied are selected as regards consanguinity with a given type of zygotes. This excludes from the beginning certain combinations of genes in the parents—no parents of RR-zygotes can have the constitution DD and vice versa, and no heterozygotes can be the offspring of marriages between the same kinds of homozygotes. But the general formulae are invalid unless all combinations can arise with the same ease.

In order to determine zygotic proportions among ancestors we must seek other openings. Let us therefore return to fig. 2 and begin with the parents of RR-zygotes. Because one of their genes always must have the character R and since the probability of the other gene's being an R-gene or a D-gene is the same as the frequency of the corresponding gene in the population, that is r and d respectively, it is evident that among, the parents concerned RR-individuals must

have the frequency r and RD-individuals the frequency d. DD-individuals obviously do not enter into the picture.

In the same manner we find that among the parents of DD-zygotes RD- and DD-individuals must have the frequencies r and d respectively. There are no RR-individuals in this connection.

Among heterozygotes' (RD) parents half must have an R-gene, half a D-gene. RR-individuals can occur only in the first half and their frequency is $\frac{1}{2}r$. Similarly the number of DD-individuals must be $\frac{1}{2}d$. RD-individuals, finally, occur in both groups and their combined frequency should be $\frac{1}{2}d + \frac{1}{2}r = \frac{1}{2}$.

Let us now pass on to the probands' grandparents. Fig. 2 discloses that half the grandparents of the RR-homozygotes have the same gene equipment as the parents, whereas the other half has the same gene make-up as the population at large. Thus, in the first half RR-individuals must have the frequency r and in the second half r^2 (vide supra). Among all grandparents the frequency of RR-individuals must therefore be $\frac{1}{2} \cdot r + \frac{1}{2} \cdot r^2 = \frac{r(1+r)}{2}$. DD-individuals, who can occur only among half the subjects, have the frequency $\frac{1}{2} \cdot d^2 \cdot \frac{(1-r)^2}{2}$, and for RD-individuals it is $\frac{1}{2} \cdot d + \frac{1}{2} \cdot 2rd = \frac{(1-r)(1+2r)}{2}$.

In a similar manner we may derive the following frequencies for grandparents of DD-zygotes:

$$\text{RR} \ldots \frac{1}{2}r^2;\ \text{DD} \ldots \frac{1}{2}d + \frac{1}{2}d^2 = \frac{(1-r)(2-r)}{2};\ \text{RD} \ldots \frac{1}{2}r + \frac{1}{2} \cdot 2rd = \frac{r(3-2r)}{2}$$

Finally, with regard to grandparents of heterozygotes (RD), fig. 2 reveals that one fourth of them have the same gene equipment as the parents of RR-zygotes, another fourth as the parents of DD-zygotes and the remaining two-fourths as the population. All parents of RD-zygotes must consequently be distributed on the three types in the following proportions:

$$\text{RR} \ldots \frac{1}{4}r + \frac{2}{4}r^2 = \frac{r(1+2r)}{4};\ \text{DD} \ldots \frac{1}{4}d + \frac{2}{4}d^2 = \frac{(1-r)(3-2r)}{4};$$
$$\text{RD} \ldots \frac{1}{4}d + \frac{1}{4}r + \frac{2}{4}2rd = \frac{1+4r(1-r)}{4}.$$

The above formulae can be obtained by proceeding in a different manner, namely by analyzing the frequencies which are given in

Table 2.

Parental combinations Type	Frequency in the population	Parents Frequency of the types RR	RD	DD	Children Frequency of the types RR	RD	DD
1.	2.	3.	4.	5.	6.	7.	8.
o. 1 RR×RR	r^4	r^4	—	—	r^4	—	—
o. 2 RR×RD	$4\,r^3\,d$	$2\,r^3\,d$	$2\,r^3\,d$	—	$2\,r^3\,d$	$2\,r^3\,d$	—
o. 3 RR×DD	$2\,r^2\,d^2$	$r^2\,d^2$	—	$r^2\,d^2$	—	$2\,r^2\,d^2$	—
o. 4 RD×RD	$4\,r^2\,d^2$	—	$4\,r^2\,d^2$	—	$r^2\,d^2$	$2\,r^2\,d^2$	$r^2\,d^2$
o. 5 RD×DD	$4\,r\,d^3$	—	$2\,r\,d^3$	$2\,r\,d^3$	—	$2\,r\,d^3$	$2\,r\,d^3$
o. 6 DD×DD	d^4	—	—	d^4	—	—	d^4
otals	$(r+d)^4 = 1$	$r^2\,(r+d)^2 = r^2$	$2\,r\,d\,(r+d)^2 = 2\,r\,d$	$d^2\,(r+d)^2 = d^2$	$r^2\,(r+d)^2 = r^2$	$2\,r\,d\,(r+d)^2 = 2\,r\,d$	$d^2\,(r+d)^2 = d^2$
			$1-r^2$			$1-r^2$	

table 2 for the different parental combinations in a population. Though that procedure is rather more complicated, it must be used in calculating the properties of collaterals. Consequently we shall deal briefly also with the latter aspect of the question.

Table 2 shows the distribution of marriage combinations in panmixia and how often the different zygotic types (columns 6–8) descend from each of the six parental combinations. It will be seen that, for example, a child of type RR can issue only from combinations 1, 2 and 4. In table 3 we have aggregated these three combinations as group A. Taken from column 6 in table 2, the frequencies in column 3 (table 3) refer to the entire population; and by dividing them with the group frequency (r^2), we have obtained the numbers in column 4 (table 3) which show the relative frequencies of every possible combination among RR-zygotes' parents. Combination no. 1 occurs only in type RR parents, combination no. 4 only in type RD parents; whereas half the parents with combination no. 2 are RR-individuals and the other half RD-individuals. Hence the frequency for this last combination must be divided equally on columns 5 and 6. The totals (r and d) in these columns consequently refer to the frequencies of types RR and RD among all parents of recessive homozygotes. The same procedure can be used to find the distribution of the different types among parents of heterozygotes (group B) and of dominant homozygotes (group C).

As we usually cannot tell RD-zygotes from DD-zygotes (both carry the dominant trait), it is of practical significance also to

Table 3.

Probands	Type	Parental combinations Frequency in the population	Parental combinations Frequency within the group	The parents of the probands Frequency of the types RR	RD	DD
1.	2.	3.	4.	5.	6.	7.
A. RR-zygotes (recessive trait-bearers)	No. 1 RR×RR	r^4	r^2	r^2	—	—
	No. 2 RR×RD	$2\,r^3\,d$	$2\,r\,d$	$r\,d$	$r\,d$	—
	No. 4 RD×RD	$r^2\,d^2$	d^2	—	d^2	—
	Totals	$r^2\,(r+d)^2$ $= r^2$	$(r+d)^2$ $= 1$	$r\,(r+d)$ $= r$	$d\,(r+d)$ $= d$	—
					$1-r$	
B. RD-zygotes	No. 2 RR×RD	$2\,r^3\,d$	r^2	$\frac{r^2}{2}$	$\frac{r^2}{2}$	—
	No. 3 RR×DD	$2\,r^2\,d^2$	$r\,d$	$\frac{r\,d}{2}$	—	$\frac{r\,d}{2}$
	No. 4 RD×RD	$2\,r^2\,d^2$	$r\,d$	—	$r\,d$	—
	No. 5 RD×DD	$2\,r\,d^3$	d^2	—	$\frac{d^2}{2}$	$\frac{d^2}{2}$
	Totals	$2\,r\,d\,(r+d)^2$ $= 2\,r\,d$	$(r+d)^2$ $= 1$	$\frac{r\,(r+d)}{2}$ $= \frac{r}{2}$	$\frac{(r+d)^2}{2}$ $= \frac{1}{2}$	$\frac{d\,(r+d)}{2}$ $= \frac{d}{2}$
					$1-\frac{r}{2}$	
C. DD-zygotes	No. 4 RD×RD	$r^2\,d^2$	r^2	—	r^2	—
	No. 5 RD×DD	$2\,r\,d^3$	$2\,r\,d$	—	$r\,d$	$r\,d$
	No. 6 DD×DD	d^4	d^2	—	—	d^2
	Totals	$d^2\,(r+d)^2$ $= d^2$	$(r+d)^2$ $= 1$	—	$r\,(r+d)$ $= r$	$d\,(r+d)$ $= d$
					1	
D. Dominant trait-bearers (RD and DD)	(No. 2–6)	$2\,r\,d + d^2$	1	$\frac{2\,r\,d\cdot\frac{r}{2}}{2\,r\,d + d^2}$ $= \frac{r^2}{1+r}$	$\frac{2\,r\,d\,(1-\frac{r}{2}) + d^2\cdot 1}{2\,r\,d + d^2}$ $= 1-\frac{r^2}{1+r}$	

calculate the frequencies for these dominant trait-carriers (RD+DD). To do so we merely have to add the expressions in columns 6 and 7 of table 3. The totals in groups B and C, on the other hand, must first be multiplied by the respective group frequencies and then divided by their sum ($2rd+d^2$), if correct figures are to be obtained. The

Table 4.

Probands: Type	Frequency in the group	Grandparents of the probands: Frequency of the types RR	RD	DD
1.	2.	3.	4.	5.
A.				
R-zygotes				
children of RR	r	$r \cdot r$	$r \cdot d$	—
children of RD	d	$d \cdot \frac{r}{2}$	$d \cdot \frac{1}{2}$	$d \cdot \frac{d}{2}$
Totals	1	$\frac{r(1+r)}{2}$	$\frac{(1-r)(1+2r)}{2}$	$\frac{(1-r)^2}{2}$
			$1 - \frac{r(1+r)}{2}$	
B.				
D-zygotes				
children of RR	$\frac{r}{2}$	$\frac{r}{2} \cdot r$	$\frac{r}{2} \cdot d$	—
children of RD	$\frac{1}{2}$	$\frac{1}{2} \cdot \frac{r}{2}$	$\frac{1}{2} \cdot \frac{1}{2}$	$\frac{1}{2} \cdot \frac{d}{2}$
children of DD	$\frac{d}{2}$	—	$\frac{d}{2} \cdot r$	$\frac{d}{2} \cdot d$
Totals	1	$\frac{r(1+2r)}{4}$	$\frac{1+4r(1-r)}{4}$	$\frac{(1-r)(3-2r)}{4}$
			$1 - \frac{r(1+2r)}{4}$	
C.				
D-zygotes				
children of RD	r	$r \cdot \frac{r}{2}$	$r \cdot \frac{1}{2}$	$r \cdot \frac{d}{2}$
children of DD	d	—	$d \cdot r$	$d \cdot d$
Totals	1	$\frac{r^2}{2}$	$\frac{r(3-2r)}{2}$	$\frac{(1-r)(2-r)}{2}$
			$1 - \frac{r^2}{2}$	
D. D- and DD-zygotes ominant trait-bearers)		$\frac{2rd \cdot \frac{r(1+2r)}{4} + d^2 \cdot \frac{r^2}{2}}{2rd + d^2} = \frac{r^2(2+r)}{2(1+r)}$	$\frac{2rd \cdot \left(1 - \frac{r(1+2r)}{4}\right) + d^2 \cdot \left(1 - \frac{r^2}{2}\right)}{2rd + d^2} = 1 - \frac{r^2(2+r)}{2(1+r)}$	

figures for parents of dominant trait-bearers that were thus obtained are entered in the last columns of the table.

The character of the grandparents of a random group of zygotes are dealt with in table 4. Among RR-zygotes' parents we have seen (table 3 group A) that type RR has the frequency r and type RD the frequency d. The first group (RR-parents) must in their turn be descended from RR-individuals and RD-individuals in the same proportions among the grandparents. Among these subjects—who as we know are grandparents of some of the probands—types RR and RD must consequently have the frequencies $r.r$ and $r.d$ respectively. On the other hand, RD-parents can be descended from individuals of types RR and RD as well as of type DD. By multiplying the frequencies concerned—which may be found in table 3 (group B)—with the relative number of RD-parents (d), we get the figures given in table 4 (under A, b) for the frequencies of these two categories of grandparents. The totals in group A thus show the zygotic proportions among all grandparents of RR-zygotes.

In view of what has been said the calculations as regards grandparents of RD-zygotes and DD-zygotes as well as of dominant trait-carriers should require no further elaboration. It will be seen that the formulae derived in tables 3 and 4 are fully identical with the formulae given above on p. 4, although the latter were obtained in a different manner.

The genetic character of the great-grandparents and even earlier ancestors can obviously be determined on similar principles.

Children and Grandchildren.

The gene frequency in the offspring of probands of a given genotype can be determined in the manner shown above for ancestors. Half the genes of known nature in first-generation offspring are transmitted from the probands, while half have come from the population via the probands' spouses and have the same character as in the population as a whole. Every succeeding generation receives a fresh contribution of genes from the population, and through this "dilution" a gene frequency approximating that in the population is eventually reached. Fig. 2 and the above formulae for the gene frequency in ancestors can be used directly for calculations concerning offspring.

To find the frequency of the various types of zygotes among the children of the probands, we again make use of table 2. Columns 3–5

in the table show the frequencies that the separate kinds of probands can attain in the possible parental combinations, and all we have to do is to distribute the frequencies concerned according to Mendel's law. Since the computations are made along the same lines as in table 3, it will suffice here to state that the frequencies obtained for zygotes' or trait-carriers' children are fully identical with the frequencies for the parents themselves, although the parental combinations are somewhat differently distributed.

With the probands' grandchildren we can deal even more briefly. It is enough to say that table 4 will do just as well for calculating the zygote proportion among grandchildren as it did among grandparents; grandchildren need only be inserted instead of grandparents and children instead of parents.

From the foregoing remarks it appears, thus, that all the formulae derived above for the genetic character of ancestral generations are valid without limitations for offspring of the corresponding generations.

Close Collaterals.

Coming now to the question of how the probands' sibs, their parents' sibs and the first cousins of (the probands) are constituted from a genetic point of view, we must note that the mere existence of sibs (and cousins) presupposes firm marital relations; free sexual relations would in practice mean that there would be half-sibs mostly. But that would not limit the applicability of simple calculus of probability.

It should be noted, moreover, that the formulae in tables 1, 2 and 3—on which are based the below calculations of the gene proportions in collaterals of certain zygotes—presuppose that all conceivable selection has been excluded so that the results will be dependent on random factors alone. Hence, in deriving formulae for the children's (i.e. the probands' sibships) probable gene proportion from the constitution of the probands' parents, these formulae will apply exclusively to children that have undergone no selection. The formulae consequently are not valid for probands selected on account of being a certain type (their nature is predetermined and not accidental); but on the contrary they do apply to their sibs whose character is due solely to accidental combinations of parental genes and goes uninfluenced by the coincidence that some probands happen to have the same parents. Probands' parents are to some extent selected too: some of their genes are defined by the choice of

probands. The formulae given below for children of grandparents to probands therefore refer not to probands' parents but to probands' parents' sibs. Similarly, our theoretical formulae for grandparents' grandchildren do not apply to the probands and their sibs but only to their first cousins.

When we determined the gene frequencies given in table 1 for probands' parents and grandparents, we simultaneously provided ourselves with the gene frequencies in the children of these ancestral generations, in other words the gene frequencies in the probands' own sibships and in their parents' sibships. For the gene proportion remains constant from one generation to the next. Consequently the formulae in column 2 of table 1 are valid for the probands' sibships just as much as for the parents, and the formulae in column 3 hold true for both grandparents and parents' sibships. The probands' first cousins are admittedly children of the parents' sibs, but cannot have the same gene frequency as the latter because they have received only half their genes from them and the other half from the population. After crossing from grandchildren to great-grandchildren, or from grandparents to great-grandparents, the dilution of the family genes proves to correspond to both the type and degree of relationship. The formulae in column 4 of the table can consequently be used also for the probands' cousins.

The frequencies of the different zygote types among the collaterals in question cannot, on the other hand, be deduced from the gene frequencies any more than could the zygote frequencies of the parents and grandparents. However, as we are now about to show, homozygotes' sibs are an exception.

In order to find the zygote frequencies in the probands' sibships, we again return to the parental combinations. Table 5, in which the computations are made, should require no comment save that the frequencies of the parental combinations (column 3), which are taken from table 3, column 4 have in columns 4–6 simply been subdivided according to Mendel's principles.

The zygote distribution in the probands' parents' sibships is shown in table 6. Table 3 contains the frequencies of zygotic types RR, RD and DD among the probands' parents. Table 5 shows, on the contrary, how often the different types occur in the sibships of all kinds of zygotes. By multiplying the numbers in question we obtain frequencies for the zygote types among parental sibs to any group of probands.

Table 5.

Probands	Parental combinations: Type	Frequency within the group	Sibs of the probands: Frequency of the types RR	RD	DD
1.	2.	3.	4.	5.	6.
A.	No. 1 RR×RR	r^2	r^2	—	—
-zygotes	No. 2 RR×RD	$2rd$	rd	rd	—
essive	No. 4 RD×RD	d^2	$\frac{d^2}{4}$	$\frac{d^2}{2}$	$\frac{d^2}{4}$
it-bearers)	Totals	1	$\frac{(1+r)^2}{4}$	$\frac{1-r^2}{2}$	$\frac{(1-r)^2}{4}$
				$1-\frac{(1+r)^2}{4}$	
B.	No. 2 RR×RD	r^2	$\frac{r^2}{2}$	$\frac{r^2}{2}$	—
)-zygotes	No. 3 RR×DD	rd	—	rd	—
	No. 4 RD×RD	rd	$\frac{rd}{4}$	$\frac{rd}{2}$	$\frac{rd}{4}$
	No. 5 RD×DD	d^2	—	$\frac{d^2}{2}$	$\frac{d^2}{2}$
	Totals	1	$\frac{r(1+r)}{4}$	$\frac{1+r(1-r)}{2}$	$\frac{(1-r)(2-r)}{4}$
				$1-\frac{r(1+r)}{4}$	
C.	No. 4 RD×RD	r^2	$\frac{r^2}{4}$	$\frac{r^2}{2}$	$\frac{r^2}{4}$
)-zygotes	No. 5 RD×DD	$2rd$	—	rd	rd
	No. 6 DD×DD	d^2	—	—	d^2
	Totals	1	$\frac{r^2}{4}$	$\frac{r(2-r)}{2}$	$\frac{(2-r)^2}{4}$
				$1-\frac{r^2}{4}$	
D.			$\frac{2rd\cdot\frac{r(1+r)}{4}+d^2\cdot\frac{r^2}{4}}{2rd+d^2}$	$\frac{2rd\cdot\left(\left(1-\frac{r(1+r)}{4}\right)+d^2\cdot\left(1-\frac{r^2}{4}\right)\right.}{2rd+d^2}$	
)- and DD-	(No. 2–6)		$=\frac{r^2(3+r)}{4(1+r)}$	$=1-\frac{r^2(3+r)}{4(1+r)}$	
;otes					
minant					
it-bearers)					

The zygote proportions in probands' cousins are derived analogously. For practical purposes these figures cannot command the same interest as the zygote frequencies for the categories of relatives

Table 6.

Probands: Type	Frequency in the group	Parents' sibs of the probands: Frequency of the types RR	RD	DD
1.	2.	3.	4.	5.
A. RR-zygotes				
a) children of RR	r	$r \cdot \frac{(1+r)^2}{4}$	$r \cdot \frac{1-r^2}{2}$	$r \cdot \frac{(1-r)^2}{4}$
b) children of RD	d	$d \cdot \frac{r(1+r)}{4}$	$d \cdot \frac{1+r(1-r)}{2}$	$d \cdot \frac{(1-r)(2-r}{4}$
Totals	1	$\frac{r(1+r)}{2}$	$\frac{(1-r)(1+2r)}{2}$	$\frac{(1-r)^2}{2}$
			$1-\frac{r(1+r)}{2}$	
B. RD-zygotes				
a) children of RR	$\frac{r}{2}$	$\frac{r}{2} \cdot \frac{(1+r)^2}{4}$	$\frac{r}{2} \cdot \frac{1-r^2}{2}$	$\frac{r}{2} \cdot \frac{(1-r)^2}{4}$
b) children of RD	$\frac{1}{2}$	$\frac{1}{2} \cdot \frac{r(1+r)}{4}$	$\frac{1}{2} \cdot \frac{1+r(1-r)}{2}$	$\frac{1}{2} \cdot \frac{(1-r)(2-r)}{4}$
c) children of DD	$\frac{d}{2}$	$\frac{d}{2} \cdot \frac{r^2}{4}$	$\frac{d}{2} \cdot \frac{r(2-r)}{2}$	$\frac{d}{2} \cdot \frac{(2-r)^2}{4}$
Totals	1	$\frac{r(1+2r)}{4}$	$\frac{1+4r(1-r)}{4}$	$\frac{(1-r)(3-r2)}{4}$
			$1-\frac{r(1+2r)}{4}$	
C. DD-zygotes				
a) children of RD	r	$r \cdot \frac{r(1+r)}{4}$	$r \cdot \frac{1+r(1-r)}{2}$	$r \cdot \frac{(1-r)(2-r)}{4}$
b) children of DD	d	$d \cdot \frac{r^2}{4}$	$d \cdot \frac{r(2-r)}{2}$	$d \cdot \frac{(2-r)^2}{4}$
Totals	1	$\frac{r^2}{2}$	$\frac{r(3-2r)}{2}$	$\frac{(1-r)(2-r)}{2}$
			$1-\frac{r}{2}$	
D RD- and DD-zygotes (dominant trait-bearers)		$\frac{2rd \cdot \frac{r(1+2r)}{4} + d^2 \cdot \frac{r^2}{2}}{2rd+d^2} = \frac{r^2(2+r)}{2(1+r)}$	$\frac{2rd \cdot \left(1-\frac{r(1+2r)}{4}\right) + d^2 \cdot (1-\frac{r^2}{2})}{2rd+d^2} = 1-\frac{r^2(2+r)}{2(1+r)}$	

discussed above, so we shall resist from making full derivations of the formulae and merely give the final results. In recessive trait-carriers' cousinships the frequency of those who carry the trait (RR) is $\frac{r(1+3r)}{4}$; in cousinships of dominant trait-carriers (RD and DD), the number of persons who carry the same trait is $1-\frac{r^2(4+3r)}{4(1+r)}$. It may be mentioned in passing that calculations of the number of trait-carriers among great-grandparents and great-grandchildren of trait-carriers lead to identical formulae.

Let us now compare with one another the zygote frequencies given in the last four tables. It is at once apparent that the formulae for parents' sibs (table 6) and those for their parents (i.e. the probands' grandparents; [table 4]) are in all respects identical, while on the other hand the formulae for probands' sibs (table 5) differ considerably from those for their parents (table 3). It is striking, moreover, that of all the types of relationships the sibships of homozygotic probands (table 5, groups A and C) are the only ones whose zygote numbers are squares, of the form $r^2+2rd+d^2$, of the gene frequencies concerned. This can be explained as follows.

The formula given above for the numerical distribution of the various zygotic types is based on an unselected population, whereas our material is selected on the basis of consanguinity with a given kind of zygotes. Fig. 2 shows how it comes about that a given proportion of the genes in the probands' ancestors—in parents two-fourths, in grandparents two-eighths etc.—always must be absolutely determined. These genes are of course those from which the probands' genes are directly derived and which therefore determine the probands' inherited constitution. In such a series of persons we must generally expect to find a gene frequency unlike that in the population from which the persons were taken, but this does not necessarily imply that certain combinations of genes are excluded or favoured, and that ratios other than those obtained by applying the binomial theorem would occur. Not only the type but also the position of certain genes are fixed in the material under discussion, and the transmissibility of these genes is in some way reduced. In parents and grandparents (as in children, grandchildren and cousins) of the probands the specific determinant genes obviously cannot be carried by the same person. They cannot possibly combine, and other combinations must therefore occur all the oftener. This means that the zygote

frequencies cannot conform to the binomial rule. By the same token the determining genes cannot meet in parents' sibs, since the genes are derived from both maternal and paternal grandparents. Because the possible combinations are equally restricted in grandparents and in parental sibs and the gene frequencies are identical, the zygote frequencies must coincide also. For similar reasons the probands' great-grandparents and cousins have the same zygote frequencies.

Things are a bit different in the probands' sibships. Their mode of arising does not rule out the possibility of two specific genes meeting; on the contrary a certain proportion of such combinations must arise. In heterozygotes' sibs, whose determinants must be D and R genes in equal proportions, three such combinations are theoretically possible: RR, RD and DD, but of these the combination RD is obviously the only one that can arise in practice[1]). This means that the genes have a restricted freedom of movement which must lead to a surplus of heterozygotes and precludes the use of the gene frequency to compute the zygotic proportion. With regard, lastly, to sibs of dominant and recessive homozygotes, there is no other conceivable combination of determinant genes than RR (or DD), and these can come into being freely so that the zygote frequencies can be obtained directly from the gene frequencies.

Remarks on the Most Important Formulae.

For practical purposes those of the above formulae are of course most important which apply to the blood relatives of recessive and dominant trait-carriers. To make things clearer these formulae have been segregated in table 7 which also gives some percentages computed on the basis of them. This makes it possible directly to read off a given trait's frequency in various types of relatives of the trait-carriers at various frequencies of the trait in the population. The mutual relations between these frequencies are shown to better advantage in figs. 3 and 4. Here the ordinates from the diagonal P represent the percentage of trait-carriers in the population while the ordinates from the other curves represent the percentage of trait-carriers in the different classes of relatives. The excess of trait-carriers among the

[1]) The sibs probably include RR's and DD's as well, but at least one of their genes must then be derived from their parents' genes, which are designated by a question mark in the pedigree.

Table 7.

in the population	Frequency of the recessive trait (RR) in the following groups of relatives of persons with the trait				in the population	Frequency of the dominant trait (RD, DD) in the following groups of relatives of persons with the trait			
	1. Sisters and brothers	2. Parents and children	3. Grandparents, grand-children and sibs of parents	4. Great-grandparents great-grandchildren, and first cousins		1. Sisters and brothers	2. Parents and children	3. Grandparents, grandchildren and sibs of parents	4. Great-grandparents, great-grandchildren and first cousins
1	2	3	4	5	6	7	8	9	10
r^2	$\frac{(1+r)^2}{4}$	r	$\frac{r(1+r)}{2}$	$\frac{r(1+3r)}{4}$	$1-r^2$	$1-\frac{r^2(3+r)}{4(1+r)}$	$1-\frac{r^2}{1+r}$	$1-\frac{r^2(2+r)}{2(1+r)}$	$1-\frac{r^2(4+3r)}{4(1+r)}$
%	%	%	%	%	%	%	%	%	%
0	25	0	0	0	0	50	50	25	12.5
0.1	26.60	3.16	1.63	0.86	0.1	50.04	50.04	25.07	12.58
0.2	27.28	4.47	2.34	1.27	0.2	50.09	50.08	25.14	12.67
0.5	28.67	7.07	3.79	2.14	0.5	50.21	50.18	25.34	12.92
1	30.25	10	5.50	3.25	1	50.44	50.38	25.69	13.34
2	32.57	14.14	8.07	5.04	2	50.88	50.75	26.38	14.19
3	34.41	17.32	10.16	6.58	3	51.32	51.13	27.07	15.03
4	36	20	12	8	4	51.76	51.52	27.76	15.88
5	37.43	22.36	13.68	9.34	5	52.20	51.90	28.45	16.73
6	38.75	24.49	15.25	10.62	6	52.64	52.29	29.14	17.57
7	39.98	26.46	16.73	11.86	7	53.07	52.65	29.82	18.41
8	41.14	28.28	18.14	13.07	8	53.52	53.04	30.52	19.26
9	42.25	30	19.5	14.25	9	53.97	53.43	31.22	20.11
10	43.31	31.62	20.81	15.41	10	54.41	53.82	31.91	20.96
12	45.32	34.64	23.32	17.66	12	55.30	54.59	33.30	22.65
14	47.21	37.42	25.71	19.85	14	56.19	55.37	34.69	24.34
16	49	40	28	22	16	57.09	56.18	36.09	26.05
18	50.71	42.43	30.21	24.11	18	57.99	56.98	37.49	27.75
20	52.36	44.72	32.36	26.18	20	58.88	57.76	38.88	29.44
25	56.25	50	37.5	31.25	25	61.15	59.81	42.40	33.70
30	59.89	54.77	42.39	36.19	30	63.45	61.90	45.95	37.97
35	63.33	59.16	47.08	41.04	35	65.76	64.01	49.51	42.25
40	66.62	63.25	51.62	45.81	40	68.10	66.20	53.10	46.55
45	69.79	67.08	56.04	50.52	45	70.46	68.43	56.71	50.86
50	72.86	70.71	60.36	55.18	50	72.86	70.71	60.36	55.18
60	78.73	77.46	68.73	64.37	60	77.75	75.51	67.75	63.88
70	84.33	83.66	76.83	73.42	70	82.81	80.62	75.31	72.66
80	89.72	89.44	84.72	82.36	80	88.09	86.18	83.09	81.55
90	94.93	94.87	92.43	91.22	90	93.70	92.40	91.20	90.60
100	100	100	100	100	100	100	100	100	100

relatives is thus shown by the vertical distance between the curves and the diagonal P.

First looking at the curves for recessive trait-carriers (fig. 3), we observe that curve I (trait-carriers' sibs) begins at 25 per cent and all the others at nil per cent. This covers the case of very rare traits (r^2 approaches zero) when both fathers and mothers of trait-carriers usually are heterozygotes and consequently will produce 25 per cent recessive homozygotes. Among the other relatives, however, we can expect heterozygotes only. With increasing proportions of trait-carriers the curves for relatives deviate quickly from the curves for the population. The excess of trait-carriers among sibs rises to 33 per cent when the frequency in the population (r^2) amounts to 12.5 per cent. The excess keeps rising among all other relatives until r^2 reaches 25 per cent. In group II (parents and children) the excess attains 25 per cent, in group III (grandparents, etc.) 12.5 per cent, and in group IV (great-grandparents, etc.) 6.25 per cent. After this maximum the curves again decline towards line P which they of course must reach if the population is composed wholly of recessive trait-carriers.

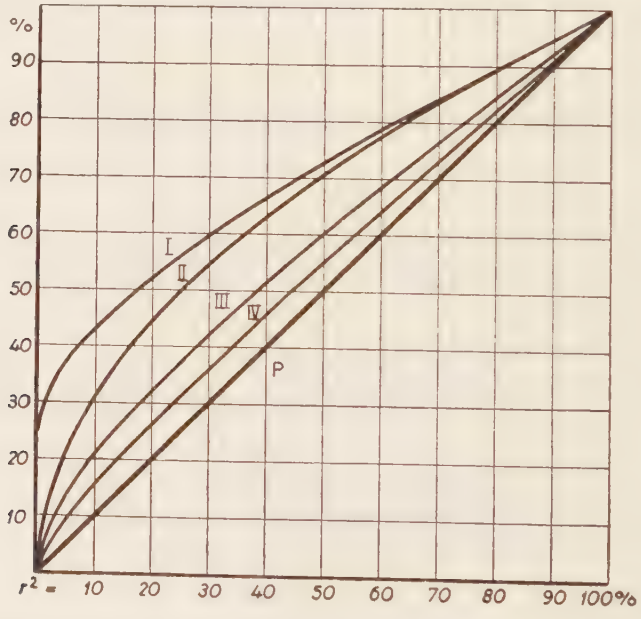

Fig. 3. Percentual number of recessive trait-bearers among the relatives of probands with the trait in question at increasing frequency of this trait in the population.

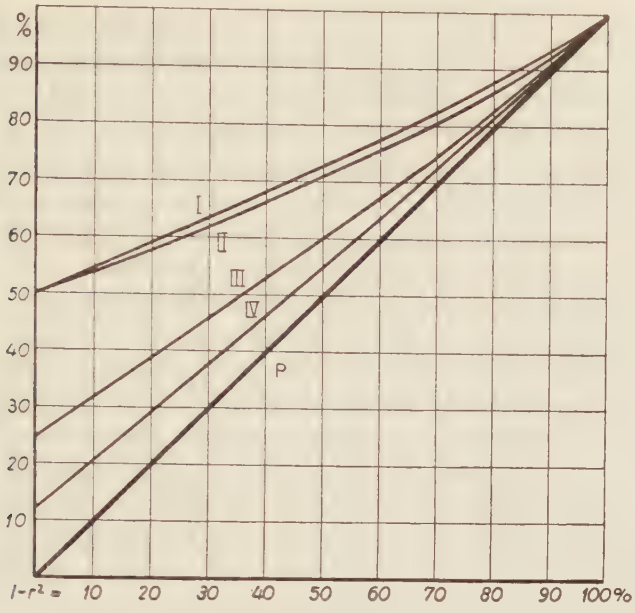

Fig. 4. Percentual number of dominant trait-bearers among the relatives of probands with the trait in question at increasing frequency of this trait in the population.

The curves for dominant trait-carriers (fig. 4) differ essentially from those for recessive. They commence at different levels: those for parents and sibs at 50 per cent, those for grandparents at 25 per cent, and those for great-grandparents at 12.5 per cent. This is easily understood considering that a dominant trait is carried by at least one ancestor in every generation, i.e. one of the parents, one of the four grandparents, one of the eight great-grandparents, etc. When a trait is very rare it is almost always carried by heterozygotes, and one out of two of their children, one out of four of their grandchildren, etc. must therefore be a heterozygous trait-carrier. The aforementioned points where the curves commence represent the maximal excess of trait-carriers among the relatives as compared with the population. From there onwards the excess gradually diminishes and the curves come ever closer together, finally meeting at the 100 per cent level. It is also noteworthy that the curves for parents and sibs lie fairly close together, the difference being 3 per cent at most. The excess of trait-carriers among grandparents (curve III) is half as large and among great-grandparents one-quarter as large as among the parents. The same, by the way, applies to a recessive trait (fig. 3).

When we finally compare the curves in fig. 3 for recessive trait-carriers with those in fig. 4 for dominant trait-carriers, we observe the following. If both types of trait-carriers, RR's and RD's plus DD's, are equally frequent in a population (50 per cent each), the two percentages will be equal in the separate classes of relatives. Then both recessive and dominant trait-carriers have a frequency of about 73 per cent in the first group and respectively of about 71, 60 and 55 per cent in the other groups. If the trait's frequency in the population exceeds 50 per cent (the right half of the figures), we will consistently find somewhat higher values when the trait is recessive than when it is dominant. But the difference is not too large, at most being about 3 per cent (group II). On the other hand, if the population contains less than 50 per cent trait-carriers, we must always expect to find more trait-carriers among the relatives when the trait is dominant than when it is recessive. The difference is greater the rarer the trait, in groups I and III coming close to 25 per cent and even passing 50 per cent in group II when it approaches zero frequency in the population. All this can also be deduced from table 7.

We shall now by a few examples illustrate how great and how characteristic is the difference both between different classes of relatives to carriers of a given trait and between dominant and recessive traits. Let the frequency of a recessive trait in a given population be 5 per cent, then its frequency among trait-carriers' relatives in group I will be about 7 times as high as in the population, in group II about 4 times as high, in group III 2 ½ times as high and in group IV almost twice as high as in the population. Another trait with the same frequency (5 %) but dominant will appear in group I just as in group II about 10 times as often in the population, almost 6 times as often in group III, and in group IV more than 3 times as often as in the population.

Suppose, on the other hand, that the general frequency of a recessive trait is 11 per cent. Carriers of the trait will then appear 4 times as often among sibs, 3 times as often among parents and children, twice as often among grandparents, grandchildren and parental sibs, and 1.5 times as often again among cousins, great-grandparents and great-grandchildren. The corresponding coefficients for a dominant trait are respectively 5, 5, 3 and 2. The relative excess of trait-carriers among relatives is slightly lower in the latter examples than in the former. In groups I and II, particularly, the excess is considerable for higher frequencies as well. The frequency is at least doubled in sibships

when the general frequency of a recessive trait does not exceed 30 per cent, and also among parents and children when it does not exceed 25 per cent. The corresponding limit in these two groups for dominant traits is about 32 per cent.

We shall see in the next and last section that these matters not only possess considerable theoretical importance, but that they in addition can be put to practical uses in genetic research.

Practical Applications of the Given Formulae.

So long as the genes remain inaccessible to direct observation, those who carry on genetic research must be content to study their manifestations as traits in the persons observed; although many traits are fashioned to a greater or lesser extent by environment. With regard to human traits, which cannot be studied by experimental mating, etc., we can only ascertain whether a given trait is hereditary or environmental by recording the presence of the trait in a selected group of related persons and observing to what extent the presence or absence of the trait agrees with the laws of heredity as found by experiments with plants and animals. We construct pedigrees and note whether the trait concerned appears more often in the families studied than in the population at large and that the frequencies do not deviate markedly from the mendelian segregation ratios. As a final measure we perhaps see whether tables can be constructed to fit the rules; this cannot seldom be done by assuming heterozygousness in certain ancestors. Where rare recessive traits are concerned we can also draw conclusions from the number of cousin-marriages among trait-carriers' parents which must be abnormally high (cf. *Dahlberg*, 1938). However, the latter method cannot be applied to ordinary traits because the excess will be unworkably small.

Whenever the given trait is rare, this method should produce fairly satisfactory results, because in such circumstances as large a preponderance of trait-carriers as would conform to the usual segregation ratios could hardly be accidental[1]).

Things are quite different when the trait to be studied occurs fairly often in the population, in let us say a few per cent of the cases.

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[1]) That one hitherto chiefly has studied rather rare characters (deformities, anomalies and other hereditary ailments) probably explains why the questions discussed in this paper have attracted but little attention in wider circles.

A purely accidental, perhaps environmental accumulation of the trait can then arise much more easily and produce the illusion of inheritance. Conceivably such a randomly increased frequency of a recessive trait might well produce a pattern resembling dominant inheritance. In the literature one encounters several instances where apparently identical traits in one pedigree appear to be recessive, in another dominant as the case may be. Quite possibly the often heard assumption is correct according to which the genes in such cases really are heterogeneous or have a weak penetrance, but in principle one should always remember that a random accumulation of trait-carriers in a family may lead to the misconception that a trait is dominant when actually it is recessive.

It should be clear from the foregoing that for statistical analyses of heredity, and particularly when the traits are at all common, we must have recourse to other and sharper methods than the mere finding that a given trait has a frequency which agrees fairly well with the classical number 1/4. Far more reliable conclusions can be drawn if one studies the frequency of the trait in each category of relatives separately and then compares the actual figures with the theoretical ones deduced from the formulae given above. Then, if all the figures show good agreement, random or environmental factors can hardly be responsible. Let us suppose, for example, that a given trait is carried by 15 per cent of a population. We proceed to analyze relatives of an adequate number of persons who have the trait themselves and encounter the trait in 48 per cent of the sibs, in 39 per cent of the parents and children, in 27 per cent of uncles and aunts and in 21 per cent of cousins of both sexes, i.e. in exactly the same proportions as in fig. 3. Then it can hardly be doubted that the trait is hereditary and, in addition, simply recessive! Dominance would be associated with quite different ratios (sibs and parents 56 per cent each, etc.; cf. fig. 4).

In the example selected, then, it is possible with some degree of certainty to decide not only that the trait is hereditary but also the type of transmission. On the other hand, if the trait has a frequency of 50 per cent it is impossible to tell whether the trait is dominant or recessive, because, as we have seen, both types of trait-carriers show the same percentages. If the trait has a still higher frequency, recessive and dominant transmission will produce frequency differences too small to warrant definite conclusions. In such cases the question: "recessive or dominant ?" cannot be answered

unless the material be analyzed in the manner described. Should a family then be found in which none of the parents have the trait, though some of their children are trait-carriers, simple dominance would of course be out of the question. Conversely, we must have simple dominance whenever no parents of an adequate number of trait-carriers lack the trait. It should be noted, finally, that the nearer the trait's frequency in the population gets to 100 per cent, the more difficult is it with the aid of our formulae and observation of the material to decide whether the trait is inherited at all, because then practically all the relatives of trait-carriers themselves have the trait. In such cases, however, we can avail ourselves of the principle of considering the rare "antagonistic" factor, i.e. the absence of the trait instead of its presence; this makes the results much clearer[1]).

If the analysis of the data produces results that disagree with the theoretical formulae, it is very likely that some sort of selection was inherent in the process used in collecting the data, and that this has influenced the frequencies. Such misadventures are the sad fate of statisticians.

If this source of error can be excluded and the trait is more common among probands' relatives than our formulae would have us expect, then environmental factors (for example contagious diseases and the like) must have entered the picture. On the other hand, if the frequencies in the families while higher than the populational means are lower than they should be theoretically, the cause must either be looked for in environmental factors or in a complicated mode of transmission. Should, finally, the frequency in the families be lower than in the population, we must again consider environment (disease control, improved hygiene, etc.).

A statistico-genealogical study of the variety indicated must be based on a fairly large mass of data. To find an adequate number of trait-carriers for probands will of course be easier the commoner the trait. But also the trait-carriers' families must be observed as thoroughly as possible, so that there is an adequate number of persons in each category of relatives. This is greatly facilitated if we can simply aggregate categories for which we have found identical formulae (e.g. parents and children; cf. table 7). Data for the pro-

[1]) If one desires to study the heredity of a pair of genes, it is usually a wise measure to pick probands from the minority. For example, if the studied factor is the colour of the eyes, we start from persons with blue eyes in southern Europe and from persons with brown eyes in northern Europe.

bands' great-grandparents and cousins rank equally in frequency calculations for relatives of group IV in the table.

A few remarks are necessary regarding the statistical analysis of data of this type. It is absolutely essential, if our theoretical formulae are to be valid, either to deal with all the trait-carriers in a population or to secure at least a representative sample of them. The probands must be selected at random irrespective of kinship or similar factors. Later some probands may prove to be married to each other and have common offspring, or some of them are sibs or otherwise related and therefore have common ancestors or collaterals. But should we while collecting the data happen to encounter probands who are so related, we must disregard any such relationships in the statistical analysis and, regarding them as fully independent cases, count their common relatives twice over (perhaps more times if several probands belong to the same family). It should hardly be necessary to mention that this principle must not be applied to "secondary cases" encountered when the families are studied in detail, and that the latter consequently must be completely segregated from the true probands.

We have seen in the foregoing that the formulae derived for probands' sibships are invalid for the probands themselves. Obviously the results of a statistical family study cannot in such circumstances be tested with these formulae unless the probands are excluded from the calculations. Here we must consequently use the well-known proband method which *Weinberg* has deduced mathematically, but from other aspects. Studies of sibs of the probands' parents and of cousins must also be done by the proband method. We must disregard the probands' parents in the first case and the probands as well as their sibs in the second, since the theoretical formulae do not cover these relationships.

After calculating the frequencies of trait-carriers in the four classes of relatives discussed above, we merely have to compare them with the corresponding theoretical frequencies in table 7 (or figs. 3 and 4). If they agree reasonably well with one another, monohybrid transmission is indicated. Such an assumption can be considered certain if, in addition, the frequency of the trait in the basic population is the same as the figure in column 1 or column 6 of the table, because this figure is the basis for all the other figures. If at first we do not know the frequency of the trait-carriers in the population, we can find out by a suitable sampling procedure. We now come to the

question of what in this connection constitutes a population. In principle a population must be limited to and comprise all persons who have an equal chance of mating with another member of the same population. Depending on the particular circumstances of the case (stationary or nomadic population, etc.) modifications are necessary in practice; no general rules can be given, however, and here as so often in statistics it must be left to the investigator's good judgment to select a control group which is as representative as possible.

Given an adequate material for statistical analysis, it luckily matters less than one would think if the limits of the population are drawn a bit too wide or a bit too narrow. If the trait is very rare one may get a considerable discrepancy between the frequency in the population of the whole country and the frequency in the population of a small district. On the other hand, if the general frequency is 1:1 000 our formulae produce practically the same results for the families of trait-carriers as if it is 1:1 000 000. Conversely, if the trait is fairly common, one should not expect that slight variations in the limits of the population would affect the frequency significantly. Besides, exact agreement is hardly necessary. The diagram shows that a small deviation in the general frequency has comparatively little effect on the family frequency.

It may be mentioned in conclusion that in applying the formulae one assumes that the trait is carried by identical genes, and that by no means always is the case (cf. *Dahlberg*, 1952). Particular attention must be directed to this aspect of the question.

The substance of this paper comes largely from an earlier publication by *Hultkrantz* and *Dahlberg* (1927) which however was published in German and therefore has been missed by English-speaking workers. That is my justification for issuing this version in English.

Summary.

In different populations you may expect different frequencies of a gene, even if panmixia can be assumed. In that case different frequencies of gene combinations are to be expected among the relatives of a proband with the trait. This problem is extensively discussed in the present paper. The main results are given in table 7. It is obvious that for investigating the inheritance of a trait you have to choose, if possible, a population where the trait is infrequent. If it is very common, of course most of the relatives have the trait

and show very small differences compared with the population. This point of view may become more important in the future, when the genetics of common traits will be investigated.

Résumé.

On peut s'attendre aux fréquences différentes d'un gène dans des populations différentes même si panmixia peut être supposé. Aussi des fréquences différentes des combinaisons de gènes doivent être attendues parmi les parents d'un cas princeps présentant le caractère. Ce problème est discuté en détail dans le travail présent. Les résultats principaux se trouvent dans le tableau 7. Il est évident que pour examiner l'hérédité d'un caractère on doit choisir, si possible, une population où le caractère est rare. Si le caractère est très commun, la plupart des parents ont le caractère et présentent des différences très insignifiantes en comparaison de la population. Cet aspect peut devenir plus important à l'avenir quand l'hérédité des caractères communs sera analysée.

Zusammenfassung.

In verschiedenen Populationen können wir verschiedene Frequenzen der Gene erwarten, selbst unter der Annahme von Panmixie. In diesem Falle können bei den Verwandten eines Probanden mit der Eigenschaft verschiedene Frequenzen der Kombination der Gene erwartet werden. In der vorliegenden Arbeit ist dieses Problem umfassend behandelt worden. Die Hauptergebnisse werden in Tabelle 7 wiedergegeben. Es versteht sich, daß zu Untersuchung der Erblichkeit einer Eigenschaft, wenn möglich eine Population ausgewählt werden muß, bei der die Eigenschaft nicht frequent ist. Ist sie von sehr gewöhnlicher Natur, besitzen selbstverständlich die meisten der Verwandten diese Eigenschaft und zeigen, verglichen mit der Population, sehr geringe Unterschiede. Dieser Gesichtspunkt wird in der Zukunft, wenn die Genetik allgemeiner Eigenschaften untersucht werden wird, mehr an Bedeutung gewinnen.

DEATH RATES IN SWEDISH PROVINCES AND THE EFFECT OF ISOLATES

By GUNNAR DAHLBERG

The Swedish people is fairly homogeneous from a hereditary point of view. There is only a small sprinkling of foreign races such as Lapps, Gipsies and Jews. These fragments of the population would not be able appreciably to affect the common death rate even if theirs differed from other Swedes'. For they are far too few to matter, with the possible exception of the Lapps who are concentrated to Northern Sweden. Perhaps more important is the fact that the population is split into more or less discrete isolates which may be slightly dissimilar. A higher rate of heterozygosis is produced by cross-overs between the isolates. It is now possible that this interchange of genes may have repercussions on the longevity of the population. Just as the increased stature in our country has been attributed mainly to isolate breaking, it is not unlikely that the same process might have produced a lower mortality and a higher longevity.

A difference in these respects could of course also be due to environmental factors, though the standard of living cannot be said to vary much from one end of the country to the other. Among the factors that next come to mind the most important is temperature, which does vary quite a lot in different parts of the country. But there is no particular evidence to show that the length of human life is affected by changes in mean temperature. We further have to consider the nature of the food, its content of vitamins and other essential substances. Obviously this is a field where variations may exist, but scarcely in any particular degree. It is nevertheless a well-known fact that the dietic habits of Northern Sweden deviate somewhat from those of Southern Sweden. While milk and cereals form the backbone of the diet in the north the people of the south mainly

subsist on animal products. Closely allied to dietic habits is the use of alcoholic beverages where there may be slight variations between one part of the country and another. But this is unlikely to make much difference with regard to mortality. Into a third group fall factors that have to do with the propagation of disease. First of all there may be geographic variations in the intensity of social intercourse and, hence, conditions may be more or less favourable for the spread of contagious diseases. Second comes the general standard of cleanliness which may vary in different parts of the country. And the last of these factors is the possibility that water infection may be more or less likely. It should also be kept in mind that the frequency of accidents, of violent deaths and of drowning may show differences regionally. The latter mortality is associated with the presence of water courses, the incidence of ability to swim, the intensity of traffic and the degree of industrialization. However, the number of deaths directly traceable to this group of factors is none too large, and dissimilarities of this type would therefore be unlikely to have a significant effect. In any case those environmental factors which are likely to affect the death rate will mainly come under the first two headings, viz. consumption of food and drink and hygiene. Here it should be recalled, however, that the mortality of elderly persons is said to be correlated to their consumption of energy and that the primary reason for the greater longevity in our country is the less heavy and exhausting nature of today's work. If this is true variations in the latter respect might of course also contribute to differences in the death rates of elderly persons. The final consideration is that there may be regional differences as regards the facilities for treating disease, i.e. in the availability of doctors and of hospital beds and equipment, and it is quite conceivable that this might have some effect on mortality. After these introductory remarks we shall now consider conditions as they actually are in different counties.

Sweden is subdivided into 25 counties, including Stockholm which ranks as a separate county for administrative purposes. The registration of deaths is probably very satisfactory. But registered deaths are not distributed equally on all age groups. A glance at figure 1 discloses that children and elderly persons have the highest mortality. Different counties cannot therefore be compared simply by equating the death rates, i.e. the number of deaths per 1000 inhabitants in any given year. For the death rate is dependent upon the age composition of the population. Suppose, for example, that a county

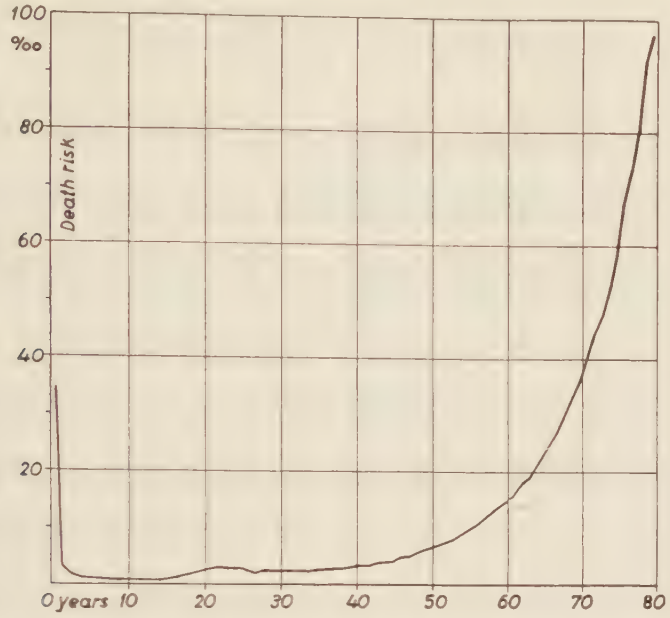

Fig. 1. Empiric death risk per 1000 men up to 80 years of age in the years 1941–45. The whole of Sweden.

has a high fertility. Then the large number of children in the county will push up the death rate to a higher level than in another county with fewer children, even if the two counties should have the same death rates age group by age group.

Although given for 5-year-groups the death rates for the different counties provide a very blurred picture of the actual state of affairs. This appears from table 1.

We have consequently calculated cumulative risks up to age 25, between 25–50 years of age and between 50–70 years of age (table 2). These figures are easier to compare. We also give diagrams of the figures for different counties for men (figures 2 and 3) and for women (figures 4 and 5).

It will be seen that the death risks for *men* up to 25 years of age are greatest in the northern counties, viz. Norrbotten, Västerbotten, Västernorrland, Jämtland and Gävleborg and on Gotland. The mortality occurring before age 25 is due to childhood mortality in a high degree. This is illustrated by a special diagram of the mortality up to 5 years of age (see figure 3). (Figures up to 1 year of age, i.e. infant mortality, are not given in official statistics for the different

Table 1. Death rates according to sex and age per 1000 individuals in different counties 31st Dec. 1945.

County	Sex	Age, years 0–5	5–10	10–15	15–20	20–25	25–30	30–35	35–40	40–45	45–50	50–55	55–60	60–70	70–ω	Total
City of Stockholm	M	9.0	1.1	0.7	1.6	2.6	2.4	2.6	3.2	4.1	8.1	11.4	17.3	33.4	97.1	10.7
	F	6.7	0.8	0.6	1.6	1.9	1.8	1.9	2.3	3.3	4.7	6.3	9.6	20.8	86.2	9.6
Stockholm	M	7.7	0.8	0.6	1.8	2.5	2.5	2.5	2.8	4.1	5.2	8.2	11.0	24.4	89.0	10.3
	F	5.3	1.0	0.7	1.9	2.6	1.9	2.0	2.4	3.0	4.0	6.8	8.5	20.5	81.1	9.6
Uppsala	M	8.3	0.9	0.3	1.6	2.3	2.5	3.0	2.9	3.7	3.9	6.8	13.4	22.4	88.8	10.6
	F	7.3	0.8	0.5	1.5	1.5	2.3	2.1	2.1	2.5	4.8	4.6	9.2	22.4	82.3	10.8
Södermanland	M	9.1	1.0	0.7	1.1	2.4	1.3	1.8	2.8	2.9	4.9	5.4	11.4	23.5	89.1	10.6
	F	6.3	0.7	0.6	1.2	1.8	1.6	1.6	2.6	2.7	5.0	8.4	12.0	23.5	89.4	11.5
Östergötland	M	9.0	1.0	0.6	1.5	1.7	1.9	2.3	2.3	2.7	4.5	8.2	11.5	23.6	92.0	10.8
	F	5.7	0.8	0.6	1.3	1.8	1.6	2.1	2.3	3.2	4.6	6.9	10.8	21.3	89.4	11.0
Jönköping	M	8.3	0.8	0.6	1.6	1.9	1.5	1.9	2.9	3.5	3.9	6.3	12.0	21.2	87.0	10.1
	F	7.1	0.5	0.5	1.7	1.9	1.2	1.8	1.6	3.3	4.4	6.3	9.8	21.2	93.4	11.0
Kronoberg	M	6.6	0.9	0.7	1.2	2.1	1.6	2.8	2.8	3.4	4.7	6.0	9.5	21.4	84.4	10.7
	F	6.6	0.7	0.6	1.8	2.1	2.3	1.5	3.2	3.2	3.8	5.9	9.8	19.2	93.3	12.2
Kalmar	M	8.8	0.9	0.9	1.4	2.9	3.2	2.3	2.8	3.3	4.2	7.5	10.4	21.5	87.5	11.0
	F	7.1	0.7	0.4	1.2	2.2	1.9	2.5	2.3	2.9	4.8	7.0	11.7	21.2	93.3	12.2
Gotland	M	8.6	0.8	0.7	3.2	5.1	5.9	4.4	3.1	4.8	6.1	8.7	9.5	23.7	89.1	12.1
	F	4.9	0.2	1.2	1.5	—	2.6	2.2	1.6	4.3	8.2	4.6	10.7	21.1	101.8	12.7
Blekinge	M	8.5	1.3	0.5	1.9	2.3	2.2	2.6	2.9	4.4	4.7	7.1	11.6	25.1	85.9	10.5
	F	6.8	0.7	1.1	1.4	3.0	2.6	2.5	2.4	3.7	5.4	5.9	11.0	18.4	87.1	11.2
Kristianstad	M	7.2	0.8	0.5	1.8	2.3	2.2	2.4	2.1	2.6	3.9	6.0	11.1	21.0	82.6	10.0
	F	6.1	0.7	0.3	1.2	2.3	1.7	2.0	2.2	3.0	4.0	5.7	9.8	19.0	83.9	10.7
Malmöhus	M	8.6	1.2	0.9	1.6	3.2	2.3	2.3	2.8	3.9	5.7	8.8	12.6	24.9	88.5	10.9
	F	6.8	0.2	0.6	1.4	1.9	2.2	2.2	2.5	3.1	4.5	6.9	9.7	20.0	87.6	10.8

Halland	M	8.7	1.3	1.2	1.5	3.3	2.4	2.1	3.2	2.3	4.3	5.6	10.5	22.9	83.5	10.5
	F	7.7	0.8	0.3	1.7	3.5	1.9	1.8	1.8	3.4	4.4	6.4	7.2	18.5	88.1	11.3
Göteborg and Bohus	M	7.3	1.0	0.6	1.7	3.0	2.1	2.5	3.1	3.8	5.3	9.0	12.2	25.0	94.7	10.1
	F	6.0	0.5	0.4	0.9	1.5	2.0	2.1	2.4	3.2	4.7	5.9	10.1	19.1	85.2	9.6
Älvsborg	M	8.8	0.9	0.5	2.1	1.4	2.0	1.9	1.9	3.8	4.9	6.8	11.2	22.4	90.3	10.6
	F	5.7	0.8	0.7	0.8	1.0	1.5	1.9	2.4	3.0	5.0	6.3	10.1	20.7	86.0	10.7
Skaraborg	M	8.2	1.0	1.2	1.5	2.4	1.5	2.4	2.6	3.1	3.8	6.4	12.1	21.0	88.6	10.9
	F	6.3	0.3	0.6	1.0	1.4	2.2	2.2	2.0	2.9	4.3	6.9	9.7	20.2	90.6	11.8
Värmland	M	8.7	1.0	0.8	0.9	2.2	1.7	2.4	2.9	3.2	5.2	8.2	12.5	24.2	95.3	11.4
	F	6.1	0.7	0.6	1.1	1.7	1.5	2.1	2.4	2.9	3.6	7.5	11.7	21.3	92.8	11.6
Örebro	M	8.8	1.1	0.9	1.5	2.4	1.9	2.2	2.2	2.4	4.5	7.2	12.0	23.0	91.3	11.1
	F	7.0	0.9	0.8	1.1	1.7	2.1	1.5	1.9	3.1	4.6	6.6	9.6	20.6	89.7	11.3
Västmanland	M	7.2	1.3	0.6	1.4	3.2	2.6	1.7	2.1	3.7	5.2	7.0	11.2	24.5	98.1	10.6
	F	6.4	0.7	0.5	1.1	1.5	1.8	1.3	1.9	2.9	4.7	5.3	9.0	22.1	87.5	10.4
Kopparberg	M	8.6	0.9	1.0	1.9	3.0	2.6	2.7	3.1	4.7	5.7	7.2	11.5	24.8	81.8	11.5
	F	6.2	0.3	0.7	0.9	2.3	1.6	1.6	2.3	2.4	4.3	7.0	10.3	22.9	98.4	11.3
Gävleborg	M	10.1	1.7	0.9	1.8	2.6	2.7	2.2	2.5	2.8	5.4	7.9	13.2	23.7	96.0	11.0
	F	8.0	0.6	1.2	1.4	1.8	2.3	1.9	2.3	3.0	4.7	7.5	10.2	21.9	91.6	10.9
Västernorrland	M	11.2	1.3	0.8	2.1	2.9	2.8	3.7	3.5	4.5	6.9	9.3	14.4	25.4	94.6	11.1
	F	8.2	1.1	0.8	1.9	2.8	2.3	3.0	2.8	4.5	5.3	7.0	10.0	24.9	98.9	11.2
Jämtland	M	9.5	1.1	0.9	2.6	3.9	3.1	3.2	3.0	3.1	4.9	5.7	12.8	23.4	93.8	10.9
	F	7.2	0.7	0.9	1.9	2.4	2.1	2.4	2.3	2.6	3.6	8.4	8.5	18.2	89.6	10.0
Västerbotten	M	10.9	1.5	1.5	2.3	3.2	2.8	2.8	2.6	3.9	5.6	7.9	11.2	23.8	96.4	9.5
	F	10.1	1.3	1.1	2.1	2.6	2.8	2.3	3.6	3.6	6.5	7.9	11.4	25.9	96.6	9.8
Norrbotten	M	12.3	1.8	1.7	2.6	4.9	3.7	3.2	3.8	4.9	6.6	9.0	15.6	29.1	95.8	10.1
	F	10.7	1.2	0.9	2.5	3.4	3.3	2.4	3.6	4.2	4.4	6.4	12.6	22.4	91.9	9.2

Table 2. Calculated cumulative death risk according to sex and between different age limits per 1000 individuals in different counties 31st Dec. 1945.

County	Age, years 0–25		25–50		50–70	
	M	F	M	F	M	F
City of Stockholm	73.9	56.5	98.3	68.5	426.8	269.3
Stockholm	65.9	56.9	82.8	64.7	315.4	264.3
Uppsala	65.8	57.1	77.2	67.6	300.8	276.2
Södermanland	70.6	52.0	66.6	66.0	298.2	311.6
Östergötland	68.4	50.4	66.3	67.0	309.7	281.1
Jönköping	64.7	57.8	67.2	59.9	282.3	274.1
Kronoberg	56.4	57.7	74.4	68.3	273.8	254.1
Kalmar	72.5	57.3	76.2	70.4	284.0	284.4
Gotland	89.8	38.3	115.6	91.4	305.0	270.3
Blekinge	70.6	64.1	81.6	80.6	319.2	251.6
Kristianstad	60.7	52.6	63.9	62.8	276.0	251.9
Malmöhus	75.9	53.7	82.1	70.6	327.5	264.7
Halland	77.4	68.0	69.7	65.6	290.4	240.0
Göteborg and Bohus	66.6	46.1	81.6	70.4	327.2	254.6
Älvsborg	67.4	45.3	70.2	67.0	292.3	270.4
Skaraborg	69.7	48.0	65.6	66.4	281.4	266.4
Värmland	66.4	50.1	74.7	61.1	318.9	287.0
Örebro	72.2	56.5	64.2	64.9	302.5	269.1
Västmanland	66.4	50.0	74.6	61.5	312.0	275.5
Kopparberg	75.2	51.1	90.3	59.9	316.9	293.9
Gävleborg	83.8	63.6	75.1	69.1	316.0	286.2
Västernorrland	89.3	72.5	102.3	86.3	340.0	311.1
Jämtland	87.3	64.1	84.0	63.3	303.3	250.1
Västerbotten	93.9	83.8	86.0	90.7	309.2	329.5
Norrbotten	112.0	90.7	106.3	86.4	375.6	295.9
The whole country	74.5	57.5	80.2	69.2	320.6	274.0

counties.) It now appears that the highest mortality exists in the northern counties but not on Gotland.

Passing on now to the mortality between the ages 25 and 50 years, we find that Gotland heads the list. Then come the northern counties of Norrbotten and Västernorrland and then the city of Stockholm. The counties of Kopparberg and Västerbotten also lie above the mean.

If we consider the mortality for the ages between 50 and 70 years it turns out that Stockholm city has a very high excess mortality which even exceeds that of Norrbotten county. Västernorrland county too has a considerable excess mortality as compared with the mean.

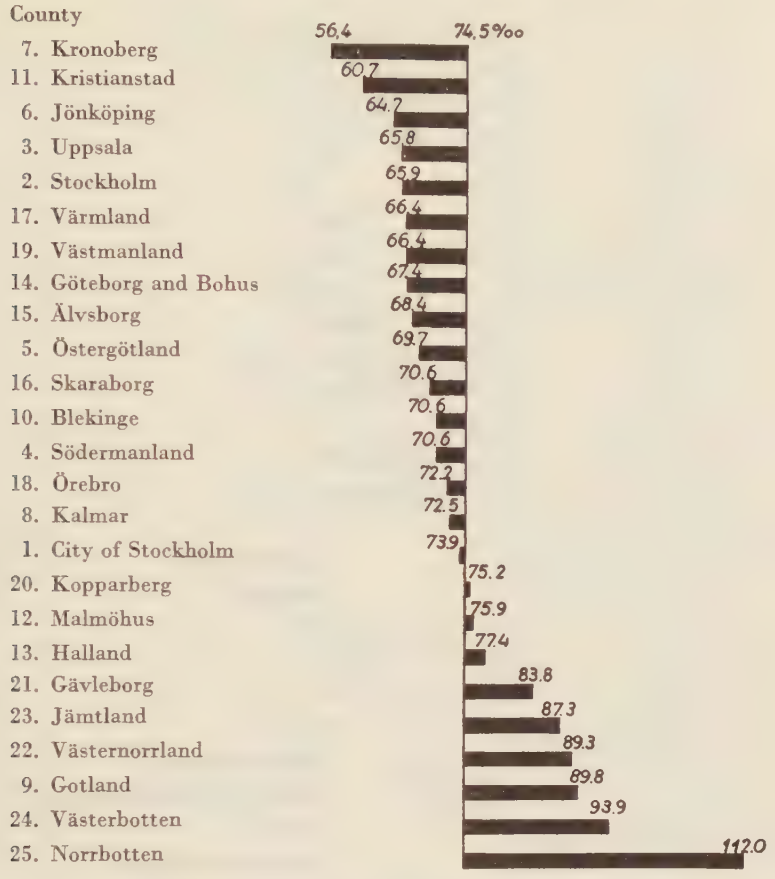

Fig. 2a. Calculated cumulative death risk per 1000 males up to 25 years of age in different counties.

That the mortality in Göteborg and Bohus county and in Malmöhus county exceeds the mean for the counties is noteworthy.

We shall now pass on to consider the risks for *women*. Up to age 25 the lead in mortality is taken by the northern counties Norrbotten, Västerbotten and Västernorrland. We also find that Holland, Blekinge, Jämtland and Gävleborg counties have figures exceeding the mean for the counties.

The figures for the age group up to 5 years (fig. 5) show that only in Norrbotten, Västerbotten and Västernorrland is the mortality really high, even if Gävleborg and Halland counties exceed the mean for the counties by a narrow margin.

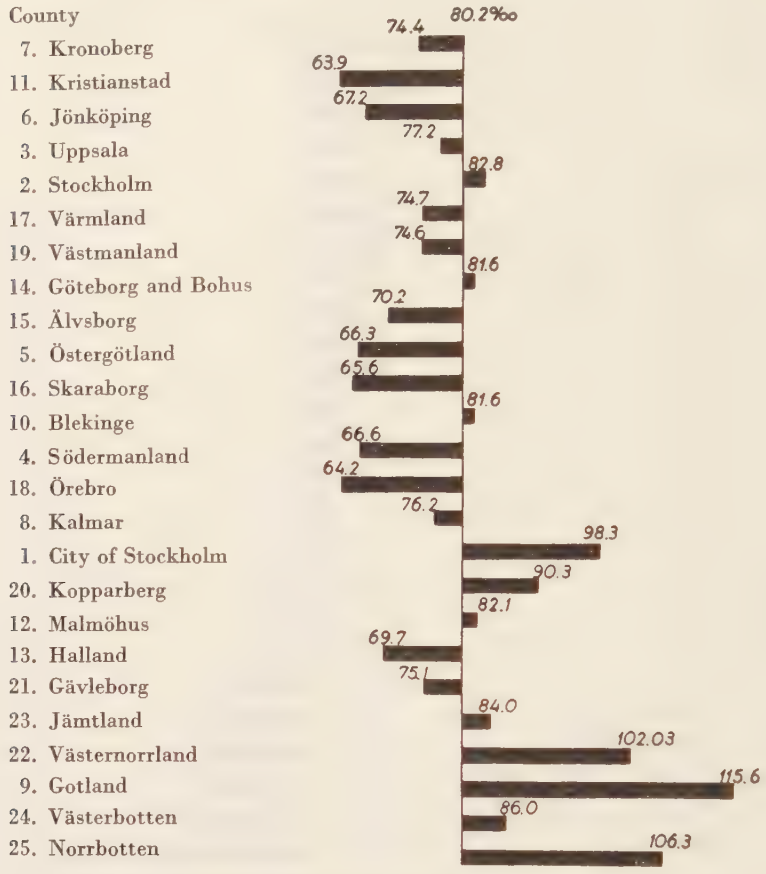

Fig. 2b (continued). Males between 25–50 years of age.

Coming now to women between 25 and 50 years of age (fig. 4), we find that high death rates are held by Gotland, chiefly, and the northern counties Västerbotten, Norrbotten and Västernorrland. Then comes Blekinge county which displays a remarkably high figure.

Let us finally consider the risks between 50 and 70 years. It is once again the three northern counties Västerbotten, Västernorrland and Norrbotten that show high figures, but so do Södermanland, Kopparberg, Värmland, Gävleborg and Kalmar counties. Östergötland county also has a remarkably high death rate.

It may here be suitable to mention that these figures cannot be used to estimate the number of old persons. Their numbers have

Fig. 2c (continued). Males between 50–70 years of age.

been calculated in a paper from the Board of Social Services by *Erland v. Hofsten* [1950]. It turns out that despite their high mortality a comparatively large number of old persons is to be expected in Norrbotten and the other northern counties. This is because death rates are not the only thing that determines the number of elderly people; the number born elsewhere and lately domiciled in the district concerned also plays an important part.

There is a real temptation to explain the figures we have found by assuming that they must be due to differences in the development of the health services in the different counties. Table 3 gives particulars with regard to the number of hospital beds. It will be seen that the number of hospital beds is relatively large in the city of Stock-

Fig. 3. Calculated cumulative death risk per 1000 males up to 5 years of age in different counties.

holm, and yet Stockholm's figures are not very impressive. Usually the mortality is higher in urban than in rural communities. In relation to the population of the county Gothenburg has few hospital beds, but its death rate is nevertheless not particularly high. Though not statistically significant, a correlation seems on the whole to exist between the number of hospital beds and the death risks for women. For men on the other hand there is a significant negative correlation between the number of individuals per hospital bed and the death rate in the 25–50 years group as well as in the 50–70 years group (cf. table 4). Put differently this means that the higher the death rate the lower is the number of hospital beds for men but not

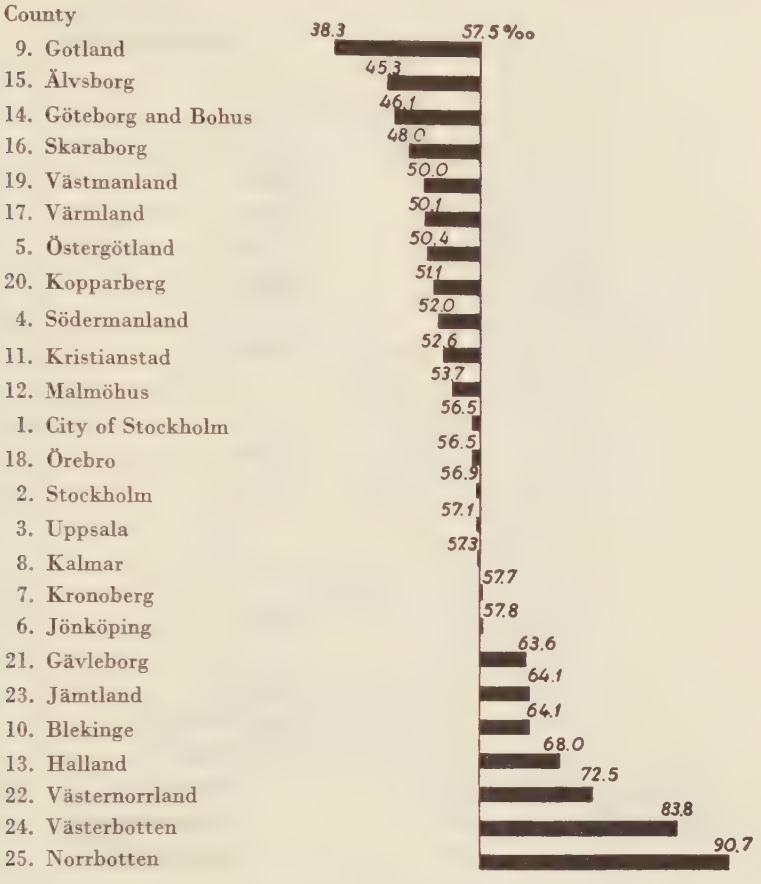

Fig. 4a. Calculated cumulative death risk per 1000 females up to 25 years of age in different counties.

for women and children. This in turn seems to indicate that the death rate has not been allowed to influence the provision of hospital beds.

The high death rate in Northern Sweden may be due to the cold climate which greatly increases the consumption of energy and therefore may indirectly create a propensity to early ageing. Worth considering is another factor, which has been mentioned previously, namely that in Northern Sweden the isolates are small and hence the incidence of intermarriages high. This might have the result that the factors which determine the length of life are especially unfavourable in the population concerned. It is obviously also true

Fig. 4b (continued). Females between 25–50 years of age.

that the state of disease control measures, particularly as regards tuberculosis, is a significant factor. In that part of Sweden, indeed, tuberculosis is most prevalent. The higher metropolitan death rate might be due to a higher consumption of energy. For a full discussion of the part played by energy consumption the reader is referred to part II of my book "Sickness and Society" (Sjukdomarna och samhället; Stockholm 1937).

With reference to the genetic effect of isolates or subpopulations it may be mentioned that the number of intermarriages will be greater if they are small than if they are large. Isolate effect serves to spread the genes and to make homozygous coincidence rarer. That intermarriages formerly were thought to have a detrimental effect

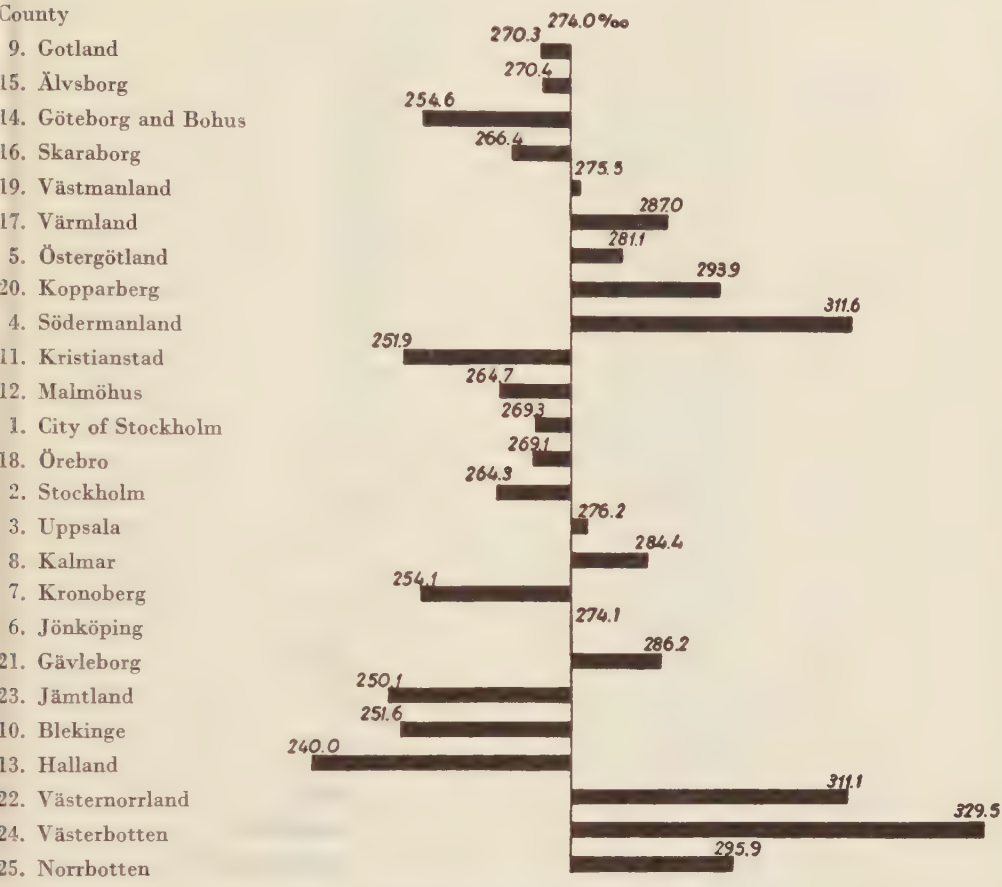

Fig. 4c (continued). Females between 50–70 years of age.

on viability and longevity is made amply clear by, for example, *Rohleder* [1912]. Already at that time he spoke of a degeneration incidental to intermarriages, and stated that intermarriages would be harmful in the long run (auf die Dauer aber schädlich sein muß). Mathematical investigations carried out by *S. Wright* [1922] on experimental animals reveal that inbreeding must result in an increased incidence of homozygotes. In the case of man *G. Dahlberg* [1947] has made theoretical studies concerning the effect of intermarriages with due regard paid to the influence of isolates, showing that the effect of intermarriage is more moderate but quite plain. Such increased homozygosis will bring together semilethal genes in double dose and thus produce an individual who dies early. Other genes,

Fig. 5. Calculated cumulative death risk per 1000 females up to 5 years of age in different counties.

such as those which determine a propensity for tuberculosis, will also meet more often in double dose. The same thing obviously applies to favourable genes, but they are irrelevant here. According to a special investigation of 17,000 schoolchildren, which was carried out in 1947, the frequency of first cousin marriages among their parents was 0.45 per cent in central and southern Sweden (cf. *Dunn* [1947]). No precise data are available for Northern Sweden as a whole, but *Böök* [1948] has found a frequency of $2.21 \pm 0.41\,\%$ in three parishes in the vicinity of Tornedalen, viz. in Pajala, Junosuando and Muonioalusta parishes. Though there is little to go on his figures indicate that the incidence of cousin marriages is higher

Table 3. Number of hospital beds in different counties.

County	No. of hospital beds	No. of individuals per hospital bed
City of Stockholm	9290	74
Stockholm	1764	191
Uppsala	1403	106
Södermanland	1359	151
Östergotland	2160	156
Jönköping	1571	165
Kronoberg	810	191
Kalmar	1383	168
Gotland	439	134
Blekinge	1160	125
Kristianstad	1222	209
Malmöhus	4956	113
Halland	1025	153
Göteborg and Bohus	4705	111
Älvsborg	2089	165
Skaraborg	1352	181
Värmland	1721	159
Örebro	1367	174
Västmanland	1208	155
Kopparberg	1936	132
Gävleborg	1696	163
Västernorrland	2027	138
Jämtland	828	172
Västerbotten	1834	125
Norrbotten	1691	136

than in the rest of Sweden, and that was to be expected. This general pattern is borne out by the lower frequency of defectives in cities than in rural parts which constitute rather small isolates, and by the markedly high frequency of defectives in Norrbotten and Väster-

Table 4. Correlation coefficients (r) between the number of individuals per hospital bed and the death rate according to sex and between different age limits.

Age, years	Males $r \pm \varepsilon (r)$	Females $r \pm \varepsilon (r)$
0 – 25	-0.30 ± 0.18	-0.12 ± 0.20
25 – 50	-0.53 ± 0.14	-0.38 ± 0.17
50 – 70	-0.70 ± 0.10	-0.21 ± 0.19

botten, i.e. in the far north of the country, despite the fact that registration in those parts would if anything tend to be more incomplete than in the rest of the country (*Dahlberg* [1947]). Analogously, the frequency of manifest factors with an effect on the survival time should be higher in these provinces.

Summary.

The variation of the death rate in different counties of Sweden is discussed. It is shown that the death rate is highest in the northern counties which may be due to the low temperature there and the consequent increase of the consumption of energy among the people. Furthermore it is pointed out that the higher death rate may also be due to the small isolates in this part of Sweden.

Résumé.

Discussion de la variation de la mortalité dans les départements différents de la Suède. Il est montré que les départements situés le plus au nord ont la mortalité la plus élevée. Cela peut dépendre du froid et de la dépense d'énergie augmentée qui en résulte pour les gens dans ces régions. En outre il est souligné que la mortalité élevée peut aussi dépendre de l'étendue insignifiante des isolats au nord de la Suède.

Zusammenfassung.

Die Variabilität der Sterbequote in verschiedenen Provinzen Schwedens wird besprochen. Es zeigt sich, daß die Sterblichkeit in den nördlichen Provinzen am höchsten ist, was auf die dortige niedrige Temperatur und den infolgedessen gesteigerten Energieverbrauch bei der Bevölkerung zurückgeführt werden kann. Weiterhin wird hervorgehoben, daß die höhere Sterblichkeit auch durch die kleinen Isolate in diesem Teil Schwedens bedingt sein kann.

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From the State Institute of Human Genetics and Race Biology, Uppsala, Sweden
(Head: Professor Gunnar Dahlberg, M.D., LL.D.)

FREQUENCY OF LONG SECOND TOE IN SWEDISH CHILDREN FROM UPPLAND PROVINCE

By TORSTEN ROMANUS

During the period 1948–1950 the relative lengths were measured of the first and second toes in a series of schoolchildren in Uppland Province. Most of them were pupils in elementary schools at Uppsala, a small proportion came from a girls' school in that town, and the rest were in elementary schools at Enköping. Furthermore, a number of male and female industrial workers from Enköping were examined.

Technique.

The subjects were instructed to place their feet one by one on a piece of paper, with the medial margin of the foot parallel to the long side of the paper and the body's weight supported by that foot. At the heel and at the tip of respectively the first and the second toe, an impression was made in the paper by means of a slender steel ruler

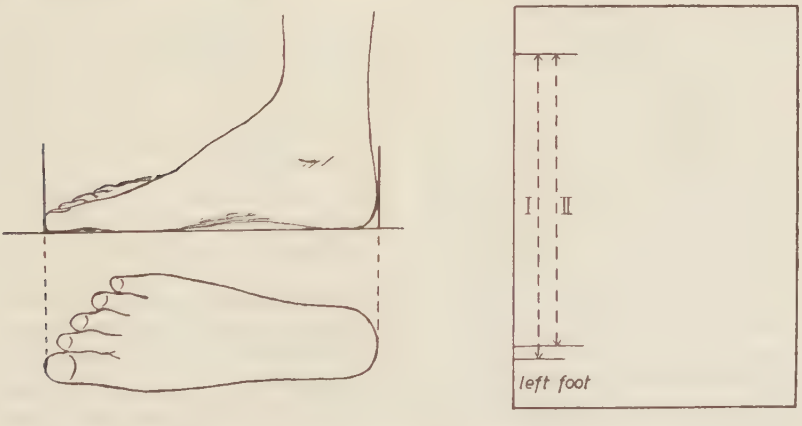

Fig. 1. Illustrates the method of measuring the foot.

held vertically (see Fig. 1). The second toe was extended if bent. On Uppsala children all measurements were taken by the author personally.

After drawing lines parallel to the short side of the paper through the "heel marks" and each of the two "toe marks", the respective distances were recorded in millimetres directly from the paper.

For the difference between the lengths of the first and the second toe the error of measurement (σ_i) was 1.1 mm.

Material.

The investigation comprised 2,307 children in elementary schools at Uppsala, aged 7–17 years, of whom 1,252 were boys and 1,055 were girls, 505 pupils in a girls' school at Uppsala, 218 children in elementary schools at Enköping (of whom 122 boys), and, finally, 320 industrial workers from Enköping, of whom 242 were males. Thus totally 3,350 individuals between 7 and 20 years of age were examined.

In tables 1 and 2 the findings have been classified by 1-year age groups and by 3 mm differences between the lengths of the first and the second toes. The median proves to fall in the 4–6 mm group, the most extreme positive difference was 19–21 mm and the greatest negative difference 10–12 mm.

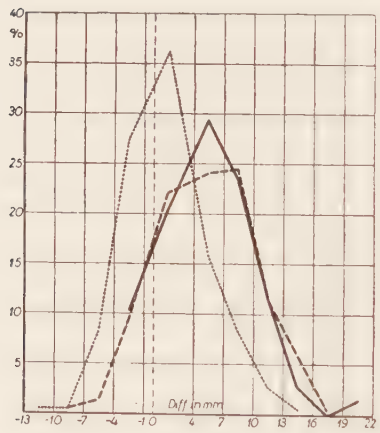

Fig. 2. Percentual distribution of individual differences (in mm) between the first and the second toe on the right foot in boys about 8 years old (dotted line), 13 years old (dashed line) and 17 years old (whole-drawn line). Cf. Table 1.

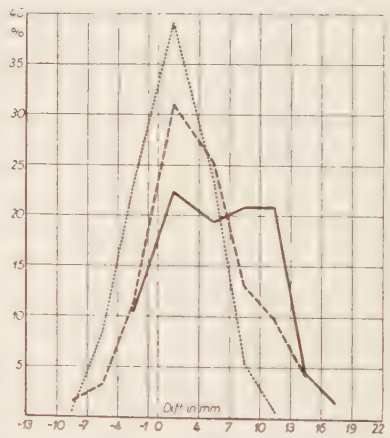

Fig. 3. Percentual distribution of individual differences (in mm) between the first and the second toe on the right foot in girls about 8 years old (dotted line), 13 years old (dashed line) and 17 years old (whole-drawn line). Cf. Table 2.

Table 1. Distribution of individual differences (in mm) between the first and the second toe on the right, respectively the left foot in boys in elementary schools at Uppsala and Enköping and in young industrial workmen at Enköping. The cases are grouped according to age.

Age, years	Difference between the first and the second toe, mm											Un-Known	Total
	Negative				0	Positive							
	10 12	7 9	4 6	1 3		1 3	4 6	7 9	10 12	13 15	16 18	19 21	
Right foot													
7					1	4	2					1	8
8		1	17	47	25	55	48	11	1			1	206
9	1	3	17	45	19	78	74	18	11	3		1	270
10		1	10	42	19	56	48	32	7	5		1	221
11		2	8	33	15	61	51	31	17	2		2	222
12			7	20	9	25	35	28	11	1	1	1	139
13		2	4	14	8	30	31	16	12	5		1	123
14			4	9	3	13	23	25	13		3	1	94
15		3	5	19	7	24	31	41	15	9	5	2	1	162
16			3	7	2	23	21	17	11	7	1			92
17				7	5	10	13	14	14	3	1			67
18				1		2	5	3	1					12
Total	1	12	75	244	113	381	382	236	113	35	11	4	9	1616
Left foot													
7				1		4	1	2						8
8	1		4	37	16	73	50	20	5					206
9		1	6	39	19	70	73	48	8	4	1		1	270
10		2	7	19	13	48	81	34	9	8				221
11			9	26	9	42	58	53	18	6	1			222
12			6	11	3	32	37	35	11	4				139
13		1	1	6	3	32	32	28	13	7				123
14			5	4	6	12	30	18	13	5	1			94
15			3	15	12	23	32	41	20	12	2		2	162
16	1			4	5	20	28	15	9	6	4			92
17			2	3	3	9	16	16	11	7				67
18						2	3	6	1					12
Total	2	4	13	165	89	367	441	316	118	59	9		3	1616

Table 2. Distribution of individual differences (in mm) between the first and the second toe on the right, respectively the left foot in girls in elementary schools at Uppsala and Enköping, in a girls' school at Uppsala and in factory-women at Enköping. The cases are grouped according to age.

Age, years	Difference between the first and the second toe, mm												
	Negative					Positive						Unknown	Total
	10–12	7–9	4–6	1–3	0	1–3	4–6	7–9	10–12	13–15	16–18	19–21	
Right foot													
7			1	1	2	4	2	3	1				14
8	1	1	15	50	3	53	29	15	5	1			183
9		1	15	32	15	54	72	28	9	1		1	228
10		3	8	31	9	48	61	39	15	3	1	2	220
11			5	23	15	48	48	28	13	4	3	1	188
12		1	6	13	4	35	48	41	32	13	3		196
13		1	3	20	6	42	52	53	25	13	1		216
14			2	3	6	24	40	39	21	11	1		147
15			1	11	2	25	34	35	16	8	3	1	136
16			2	6	1	15	23	24	15	7	1		94
17				7	2	12	20	16	8	2		1	68
18				1		6	9	6	3	2	1	1	29
19				1			3	3	2	2	1		12
20								1	2				3
Total	1	7	58	199	75	366	441	331	167	67	15	3	4	1734
Left foot													
7			1		1	5	4	3					14
8			9	26	17	55	50	19	4	3			183
9			6	27	8	63	62	48	11	2	1		228
10			4	17	12	44	63	45	28	6	1		220
11			2	8	10	45	57	37	19	7	2	1	188
12			3	11	3	24	46	53	32	17	6	1	196
13			3	9	11	40	44	57	32	15	2	3	216
14				6	4	23	24	25	32	12	1		147
15			1	4	5	17	40	36	16	10	7		136
16				7	3	12	25	23	12	9	3		94
17				3	3	13	21	13	14		1		68
18				1		5	5	10	5	2	1		29
19				1		1	1	3	5		1		12
20							1	2					3
Total			29	120	77	347	443	394	210	83	26	1	4	1734

Table 3. Percent extreme positive (> 10 mm) and negative (> 4 mm) differences between the first and the second toe in the total number of cases, grouped according to age and sex.

Age, years	Right foot					Left foot				
	Total No. of diff.	Pos. difference (> 10 mm)		Neg. difference (> 4 mm)		Total No. of diff.	Pos. difference (> 10 mm)		Neg. difference (> 4 mm)	
		No.	Per cent	No.	Per cent		No.	Per cent	No.	Per cent
	Boys									
7	7					8				
8	205	1	0.5	18	8.8	206	5	2.4	5	2.4
9	269	14	5.2	21	7.8	269	13	4.8	7	2.6
10	220	12	5.5	11	5.0	221	17	7.7	9	4.1
11	220	19	8.6	10	4.5	222	25	11.3	9	4.1
12	138	14	10.1	7	5.1	139	15	10.8	6	4.3
13	122	17	13.9	6	4.9	123	20	16.3	2	1.6
14	94	17	18.1	4	4.3	94	19	20.2	5	5.3
15	161	31	19.3	8	5.0	160	34	21.3	3	1.9
16	92	19	20.7	3	3.3	92	19	20.7	1	1.1
17	67	18	26.9			67	18	26.9	2	3.0
18	12	1	(8.3)			12	1	(8.3)		
Total	1607	163	10.1	88	5.5	1613	186	11.5	49	3.0
	Girls									
7	14	1	(7.1)	1	(7.1)	14			1	(7.1)
8	183	6	3.3	17	9.3	183	7	3.8	9	4.9
9	227	10	4.4	16	7.0	228	14	6.1	6	2.6
10	218	19	8.7	11	5.0	220	35	15.9	4	1.8
11	187	20	10.7	5	2.7	187	28	15.0	2	1.1
12	196	48	24.5	7	3.6	196	56	28.6	3	1.5
13	216	39	18.1	4	1.9	213	49	23.0	3	1.4
14	147	33	22.4	2	1.4	147	45	30.6		
15	136	28	20.6	1	0.7	136	33	24.3	1	0.7
16	94	23	24.5	2	2.1	94	24	25.5		
17	68	11	16.2			68	15	22.1		
18	29	7	24.1			29	8	27.6		
19	12	5	(41.7)			12	6	(50.0)		
20	3	2	(66.7)			3				
Total	1730	252	14.6	66	3.8	1730	320	18.5	29	1.7
	Both sexes									
7	21	1	4.8	1	4.8	22			1	4.5
8	388	7	1.8	35	9.0	389	12	3.1	14	3.6
9	496	24	4.8	37	7.5	497	27	5.4	13	2.6
10	438	31	7.1	22	5.0	441	52	11.8	13	2.9
11	407	39	9.6	15	3.7	409	53	13.0	11	2.7
12	334	62	18.6	14	4.2	335	71	21.2	9	2.7
13	338	56	16.6	10	3.0	336	69	20.5	5	1.5
14	241	50	20.7	6	2.5	241	64	26.6	5	2.1
15	297	59	19.9	9	3.0	296	67	22.6	4	1.4
16	186	42	22.6	5	2.7	186	43	23.1	1	0.5
17	135	29	21.5			135	33	24.4	2	1.5
18	41	8	19.5			41	9	22.0		
19	12	5	(41.7)			12	6	(50.0)		
20	2	2	(66.7)			3				
Total	3337	415	12.4	154	4.6	3343	506	15.1	78	2.3

Table 4. Mean differences [D ± ε (D)] in mm between the first and the second toe in the examined cases. The different groups of individuals are divided according to sex and age.

Age, years	Boys				Girls				Girls			
	Right foot		Left foot		Right foot		Left foot		Right foot		Left foot	
	No.	D ± ε (D)	No.	D ± ε (D)	No.	D ± ε (D)	No.	D ± ε (D)	No.	D ± ε (D)	No.	D ± ε (D)
	Elementary schools in Uppsala								*Girls' school in Uppsala*			
7	7	2.6	8	3.3	14	3.5	14	3.6				
8	205	1.8±0.22	206	2.8±0.22	183	1.9±0.28	183	3.1±0.26				
9	217	2.3±0.25	217	3.2±0.22	185	2.8±0.26	186	3.7±0.25				
10	172	2.6±0.28	173	3.7±0.27	177	3.1±0.30	179	4.6±0.28				
11	198	3.4±0.28	200	4.5±0.25	167	3.9±0.38	167	5.2±0.28	7	5.9	7	5.0
12	138	4.3±0.37	139	4.9±0.34	107	5.5±0.41	107	5.8±0.43	89	7.9±0.43	89	8.6±0.45
13	121	4.2±0.40	122	5.5±0.37	113	5.1±0.39	110	5.5±0.36	103	6.3±0.46	103	7.0±0.45
14	63	4.4±0.55	63	5.0±0.55	48	5.6±0.66	48	6.6±0.61	87	7.1±0.38	87	7.8±0.40
15	101	4.1±0.52	100	5.4±0.46	11	5.2±0.67	11	6.6±0.57	77	6.3±0.49	77	7.0±0.48
16	20	4.3±1.27	20	5.3±0.95	12	5.0±(1.04)	12	4.5±(1.27)	59	6.4±0.61	59	6.8±0.66
17	1		1		1		1		41	4.9±0.72	41	5.4±0.64
18									27	6.5±0.89	27	7.1±0.85
19									12	8.8	12	8.7
20									3	9.3	3	7.7
	Elementary schools in Enköping											
9	52	4.4±0.52	52	6.8±0.78	42	5.1±0.59	42	6.3±0.60				
10	48	5.4±0.57	48	6.6±0.57	41	6.4±0.61	41	8.4±0.47				
11	22	5.3±0.76	22	6.1±0.58	13	7.6	13	7.8				
	Industrial undertakings in Enköping											
13	1		1									
14	31	8.3±0.78	31	7.1±0.71	12	6.3±(0.42)	12	6.0±(1.30)				
15	60	7.5±0.66	60	6.9±0.55	15	8.6±1.20	15	8.0±1.58				
16	72	5.9±0.50	72	6.4±0.45	23	6.8±0.61	23	7.9±0.85				
17	66	5.8±0.63	66	6.7±0.56	26	6.6±0.54	26	6.6±0.69				
18	12	5.2±(0.94)	12	6.5±(0.50)	2		2					

Table 5. Mean differences [D ± ε (D)] in mm between the lengths of the right and the left foot. All examined cases distributed according to age and sex.

Age, years	Boys			Girls		
	No.	D ± ε (D)	σ	No.	D ± ε (D)	σ
7	8	—3.50		14	—1.54	
8	205	—1.91±0.26	3.78	183	—1.73±0.26	3.57
9	268	—1.78±0.31	5.04	228	—2.21±0.29	4.45
10	220	—1.95±0.30	4.50	220	—1.99±0.34	5.00
11	218	—2.35±0.26	3.80	187	—1.56±0.27	3.68
12	137	—1.68±0.32	3.79	196	—1.76±0.27	3.76
13	123	—2.83±0.39	4.30	216	—0.79±0.27	3.92
14	93	—0.77±0.50	4.77	147	—0.29±0.41	4.94
15	159	—2.42±0.32	3.98	136	—0.64±0.33	3.86
16	92	—1.39±0.42	4.00	94	—0.16±0.35	3.39
17	67	—1.40±0.40	3.30	68	+0.93±0.59	4.86
18	12	—1.50		29	—0.10±0.51	2.76
19				12	+0.67	
20				3	+8.00	

Curves for the ages 8, 13 and 17 years are given in figures 2 and 3, which reveal that there tend to be more positive differences between the lengths of the first and second toes at higher age levels. The second toe of younger children is in other words longer relatively more often, and they also on the whole show a greater negative and a smaller positive difference between the first and the second toe.

A similar pattern can be distinguished in table 3, showing the frequencies in several age groups of extreme positive differences (10 mm and up) and of marked negative differences (4 mm and up) between the first and the second toe. Extreme positive differences in the 7–20 years category occurred in about 12 per cent of the children with regard to the right foot and in about 15 per cent with regard to the left. The frequency of extreme differences for the right foot in the 7–9 years group of children was only about 5 per cent, among the 10–11 year-olds 8–10 per cent and between 12 and 18 years of age around 20 per cent. The frequency of extreme negative differences declines similarly: 9–8 per cent in the 8–9 year-olds, 5–4 per cent in the 10–12 year-olds and about 3 per cent in the 13–16 year-olds. For the left foot things were much the same. There were no differences between boys and girls in this respect.

Regarding the mean differences (table 4) between the first and the second toe at different age levels in elementary school pupils at Uppsala, one finds in the 8–11 years group a difference of about 2–4 mm for the right foot, while for the left it is somewhat larger, namely 3–5 mm. In the 12–16 years group the left foot showed a

Table 6. Distribution of the differences between the lengths of the same individual's right and left foot. All cases.

	Difference, mm	Boys		Girls		Both sexes	
		No.	Per cent	No.	Per cent	No.	Per cent
Positive differences	28 –30			1	0.06	1	0.03
	25 –27						
	22 –24	1	0.06			1	0.03
	19—21	1	0.06	1	0.06	2	0.06
	16 –18			1	0.06	1	0.03
	13 –15	3	0.19	2	0.12	5	0.15
	10—12	4	0.25	6	0.34	10	0.30
	7— 9	13	0.81	35	2.02	48	1.44
	4— 6	92	5.74	136	7.84	228	6.84
	1— 3	287	17.92	393	22.68	680	20.39
	0	165	10.30	196	11.31	361	10.82
Negative differences	1— 3	494	30.84	507	29.25	1001	30.01
	4— 6	332	20.72	298	17.20	630	18.89
	7— 9	151	9.43	106	6.12	257	7.71
	10—12	40	2.50	33	1.90	73	2.19
	13 –15	11	0.69	8	0.46	19	0.57
	16—18	4	0.25	7	0.40	11	0.33
	19—21	2	0.12	1	0.06	3	0.09
	22 –24	1	0.06			1	0.03
	25—27	1	0.06	1	0.06	2	0.06
	28 –30			1	0.06	1	0.03
	Total	1602	100	1733	100	3335	100

difference of 4–5 mm and the right 5–6 mm. Neither here, there were any dissimilarities between boys and girls. It stands to reason that the mean difference must increase with age; the absolute length of the foot increases proportionately. The fact that the left foot's mean difference is bigger fits in well with the above.

Comparing now the right and left foot, one finds extreme positive differences on 15.14 per cent of the left feet (boys and girls) but on 12.44 per cent of the right feet, the difference being 2.70 ± 0.83 per cent, and for girls separately 3.93 ± 1.26 per cent and thus statistically significant. For boys separately on the other hand the difference is merely 1.39 ± 1.06 per cent. Similarly, the frequency of extreme negative differences is lower on the left foot in all age groups and diminishes with increasing age.

Table 5 discloses that the left foot on an average is about 2 mm longer than the right foot. This applies to boys from 8 to 18 years of age and to girls between 8 and 14 years old. Girls over 14 seem to have a slightly smaller mean difference.

In table 6 the material is classified in regard to the difference between the same child's two feet. It will be seen that about 10 per

cent of the children had feet of equal length, while about 60 per cent of them had the left foot longer than the right, the difference exceeding 6 mm in about 11 per cent and 9 mm in about 3 per cent. Maximal differences between the right and the left foot were between 20 and 30 mm.

Summary.

The practical implications of this study would seem to be that shoes for young children should be designed to allow more space for the second toe; that the fit of a new pair of shoes first should be tried on the left foot which usually is bigger than the right; and, perhaps, in extreme cases special shoes should be used.

Zusammenfassung.

Der praktische Gehalt dieser Studie ergibt sich aus der Forderung, daß Schuhe für Kinder niederer Altersklassen eine Ausführung besitzen sollen, welche der zweiten Zehe mehr Platz gewährt – daß ein paar neuer Schuhe zuerst an dem linken Fuße, welcher gewöhnlich größer als der rechte ist, angepaßt werden sollte – und daß unter Umständen in extremen Fällen speziell angefertigte Schuhe getragen werden sollten.

Résumé.

La signification pratique de cette étude semble être que les chaussures pour des enfants doivent être faites d'une manière qu'elles laissent plus de place pour le deuxième orteil; que des chaussures neuves doivent être essayées premièrement au pied gauche qui est en général plus grand que le pied droit; et, peut-être, que des chaussures spéciales doivent être employées dans des cas extrêmes.

From The State Institute of Human Genetics and Race Biology, Uppsala, Sweden.
(Head: Professor Gunnar Dahlberg, M.D., LL.D.)

A NOTE ON THE RELATIVE LENGTH OF THE NOSE

Nose-Lip Index in the Bernadottes

By TORSTEN ROMANUS, M.D.

The central position of the nose makes it a characteristic structural feature of the face. That is why anthropologists systematically have studied its shape, its length and breadth and the ratio between the two. But less attention has been paid to the relations between the nose and other parts of the face. Anthropology has on the whole concerned itself less with the soft tissues than with those parts of the skeletal framework that can be measured. There is nothing strange about this however—in the soft tissues it is difficult to find fixed points to measure from. Unless they are made from points exactly defined, measurements will not be comparable.

Here we shall consider the ratio between the shortest distance from the medial corner of the eye (angulus medialis oculi) to the cranial border of the mucous membrane of the upper lip and the length of the nose. It would seem that this ratio has not been studied and that the inner canthi has not been employed as the proximal point of reference. A few remarks on the measuring technique may therefore be in place. Since the proximal point for measuring the length of the nose—usually the nasion (see for example: A. Hrdlicka: "Anthropometry", Philadelphia, 1920)—may be difficult to fix, the corner of the eye was used instead as the proximal point for the purposes of this investigation. The corresponding distal point was the root of the wing of the nose (punctum subnasale alae nasi) on the same side. The straight line joining these points is as a rule parallel to the midline of the face. The point of reference on the upper lip lies at the intersection of the proximal border of its mucous membrane and the line trough the corner of the eye and the root of the alae nasi.

Let the point at the corner of the eye, that at the root of the alae nasi and that at the upper lip be called O, N and L respectively. If we now measure the length of ON, divide it by the length of OL (the measured person's mouth should of course be closed), and multiply the result by 100, we obtain an index which here is called the nose-lip index. Thus, $\frac{ON}{OL} \cdot 100$. Indirectly this ratio provides a measure of the "height" of the upper lip. If there is no upper lip and the nose occupies all the distance between the corner of the eye and the mouth, the index equals 100. One might say that this is the case in predatory animals. Again, if there is no nose, the index is 0, as in certain birds. An index of 50 implies that the nose is relatively short, reaching only halfway to the margin of the upper lip.

Let us now consider the nose-lip index in man. How large is it?

The following figures were obtained by taking measurements on 106 randomly selected Swedes, students of medicine and dentistry in their twenties, among whom 51 were men and 55 women.

Index		Men	Women	Cases
	60	0	1	1
	61	2	0	2
	62	1	3	4
	63	1	2	3
	64	5	1	6
	65	5	5	10
	66	3	6	9
	67	14	9	23
	68	8	7	15
	69	4	8	12
	70	1	6	7
	71	0	3	3
	72	5	3	8
	73	1	0	1
	74	1	1	2
Totals		51	55	106
Mean Index:		67.2	67.5	67.3

In old age the face is furrowed and the mouth sunken. The nose-lip index must therefore be expected to rise somewhat as the years go by.

The nose-lip index can be used as a racial character. Negroes and Mongols would have a low index while the Mediterranean peoples ought to have a relatively high index. It will be seen from the table, however, that the nose-lip index varies within one and the same population.

No profile measurement, the nose-lip index lends itself to photographic measuring. It is best to work with frontal projections; though it should be noted that the projection should not be oblique in the vertical plane, as that would distort the readings.

* * *

A low nose-lip index can be characteristic of the facial features in certain families. As will be shown in the following there are grounds for supposing that a low nose-lip index can be inherited as a simple dominant character. The Bernadotte Dynasty, the Reigning Royal Family in Sweden, may be taken as an example of this. In analogy with the House of Habsburg's famed lower lip one might call this trait "the Bernadottian upper lip".

For the following survey of the nose-lip index in the House of Bernadotte the measurements were taken partly from picture post cards available commercially, partly from pictures in the archives of the "Svenska Dagbladet" and of Messrs. Åhlen & Åkerlund, partly from portraits in the archives of the Royal Gallery and at the Royal Palace, Stockholm. The figures below the names in the genealogy represent the nose-lip index. Figures in parentheses are very approximate, either because the pictures were small or the projections oblique. Many of the figures were derived from many different photographs. In such cases the various measurements agreed remarkably well.

A glance at the genealogy reveals that the nose-lip index is below 60 in the line Royal for four generations from King Gustavus V. Among King Gustavus's brothers Prince Oscar Bernadotte and his son Folke Bernadotte and grandson Folke Bernadotte Junior also have low nose-lip indices. So have King Gustavus's son Prince Wilhelm and the latter's son Lennart Bernadotte. The same holds true for Prince Bertil and for Sigvard and Carl Johan Bernadotte.

The early generations Bernadotte and the progenitor Karl Johan all have an index that lies over or close to the mean. (Among the female members of the Dynasty Queen Ingrid and the Princess Christina have an index of 62 which perhaps should be considered

low.) It is said that King Gustavus was the image of his mother, Queen Sofia of Nassau. And it is in her that we first find a low nose-lip index. On available photographs it is as low as 56. The characteristic Bernadottian high upper lip probably derives from Queen Sofia. Pictures suitable for measuring on Queen Sofia's parents have not been accessible. Queen Sofia's father, Prince Wilhelm of Nassau, may have a nose-lip index of approximately 62. It is possible, however, that the gene is due to a mutation.

It has been possible to compare pictorial measurements with measurements taken directly on the person concerned for one branch of the family. These direct measurements were taken by Mr. Folke Bernadotte Junior, who is at present studying medicine, and on him by the author.

	Nose-Lip Index:	
	Photographic	Direct
Prince Oscar Bernadotte	61	60
The Countess Estelle Bernadotte . .	64	63
Mr. Folke Bernadotte Junior . . .	61	61
Mr. Bertil Bernadotte II	(65)	67

It will be seen that the two sets of figures are remarkably alike.

It may be mentioned that a low nose-lip index also occurs in the House of Windsor. Such is the case for example with Queen Victoria and the Duke of Windsor. This trait too might possibly derive its origin from a German progenitor.

Since a low nose-lip index seems to be inherited as a dominant trait, it might find a use as an auxiliary paternity test.

The illustrations on the following pages are diminutions of the photographs on which the measurements were made.

I

Fig. 1. Karl XIV Johan
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II

Fig. 2. Oscar I
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III

Fig. 3. Oscar II
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Fig. 4. Sofia
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IV

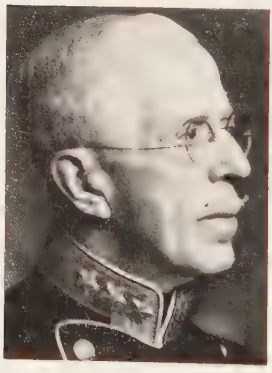

Fig. 5. Gustavus V
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Fig. 6. Viktoria
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Fig. 7. Oscar B.
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Fig. 8. Carl senior
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Fig. 9. Eugen
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Fig. 10. Gustavus VI A.
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Fig. 11. Wilhelm
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Fig. 12. Folke B.
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Fig. 13. Estelle B.
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Fig. 14. Gustavus A.
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Fig. 15. Sigvard
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Fig. 16. Bertil
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Fig. 17. Carl Johan
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Fig. 18. Folke B. jr.
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Fig. 19. Margaretha
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Fig. 20. Birgitta
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Fig. 21. Desirée
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Fig. 22. Christina
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Fig. 23. Carl Gustavus
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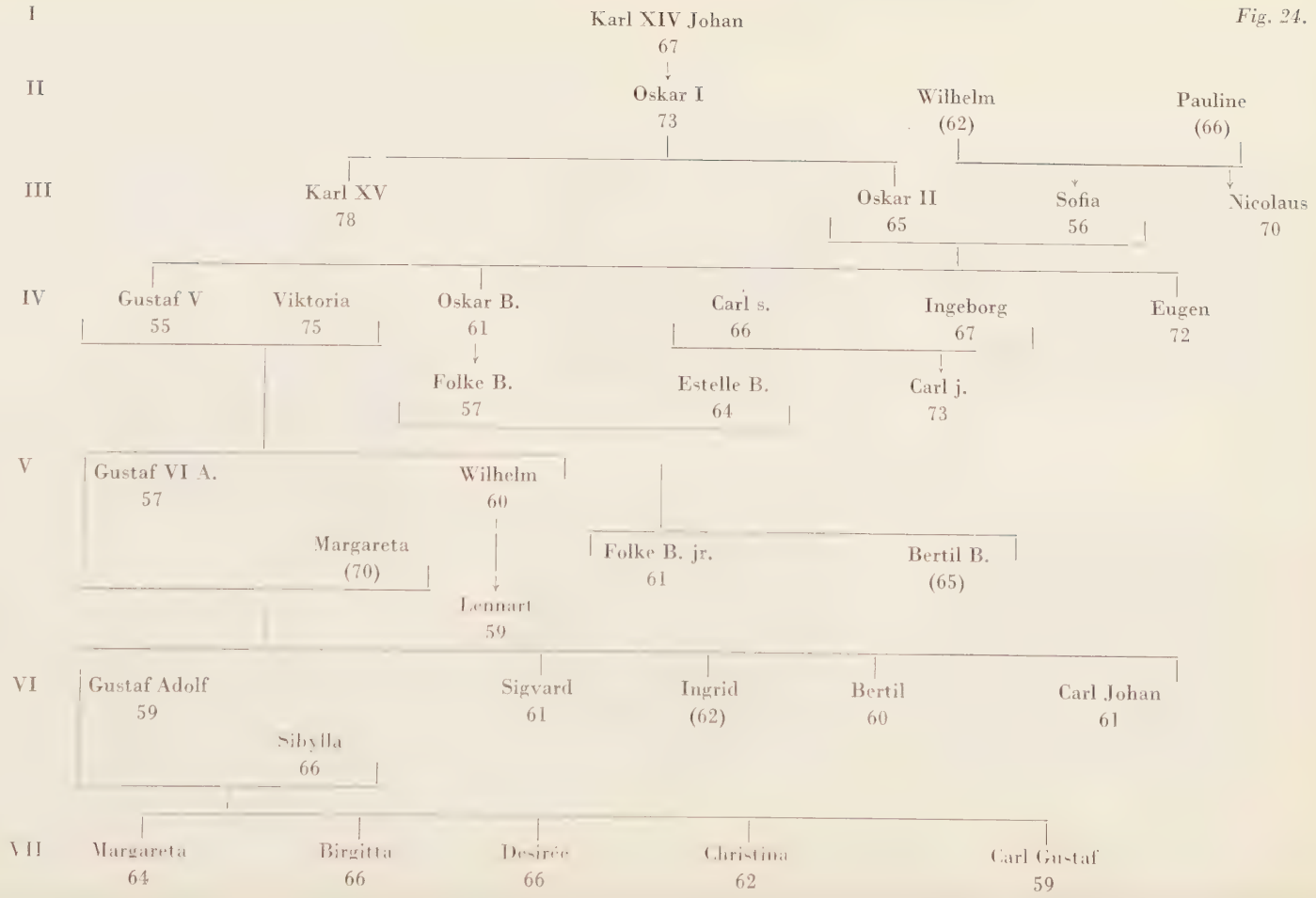
Fig. 24.


I
Karl XIV Johan
67
II
Oskar I
73
Wilhelm
(62)
Pauline
(66)
III
Karl XV
78
Oskar II
65
Sofia
56
Nicolaus
70
IV
Gustaf V
55
Viktoria
75
Oskar B.
61
Carl s.
66
Ingeborg
67
Eugen
72
Folke B.
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Estelle B.
64
Carl j.
73
V
Gustaf VI A.
57
Wilhelm
60
Margareta
(70)
Folke B. jr.
61
Bertil B.
(65)
Lennart
59
VI
Gustaf Adolf
59
Sigvard
61
Ingrid
(62)
Bertil
60
Carl Johan
61
Sibylla
66
VII
Margareta
64
Birgitta
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Desirée
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Christina
62
Carl Gustaf
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Aus dem Anthropologischen Institut der Universität München
(Direktor: Prof. Dr. Dr. K. Saller)

BESTEHEN BEZIEHUNGEN ZWISCHEN DEM INDIVIDUELLEN QUANTITATIVEN WERT IM HAUTLEISTENSYSTEM UND DEN BLUTGRUPPEN?

Von HELMUT BAITSCH

Mit der von *Landsteiner* und *Wiener* entdeckten Blutkörpereigenschaft Rh hat die Frage der heterospezifischen Schwangerschaft, d.h. der Verschiedenheit von Mutter und Kind in ihren Bluteigenschaften (als Pathogenese von *Dienst* schon 1905 vermutet) erneut, vor allem klinische, Bedeutung erlangt. Inzwischen weiß man, daß es bei fast allen Blutgruppensystemen zu Antigen-Antikörper-Immunreaktionen und damit zu vielfältigen Schädigungen der Frucht kommen kann.

Trotz der Häufigkeit von heterospezifischen Schwangerschaften treten nur wenig schwere Erkrankungen auf. Dies mag u.a. damit zusammenhängen, daß geschädigte Früchte schon früh ausgestoßen werden (*Loghem* und *Spaander*, *Waterhous* und *Hogben*), bzw. in anderen Fällen das Kind die mütterlichen Antikörper abbindet und unschädlich macht (*Bloksom* und *Matthaei*). Für ein absolut häufigeres Vorkommen sprechen die Untersuchungen von *Halbrecht:* Bei 200 normalen Kindern bestand in 26,5 % A-B-0-Heterospezifität; sie fand sich jedoch in 95 % (bei *Wiener* in 81 %) der Fälle von Icterus neonatorum praecox ("simulating mild erythroblastosis"). *Soulairac* u.a. diagnostizierten bei debilen Kindern einen hohen Prozentsatz von Inkompatibilität mit ihren Müttern speziell im A-B-0-System und den MN-Faktoren; im Rh-System war der Prozentsatz gegenüber der Erwartung nicht erhöht. In früheren Arbeiten hatten *Hirszfeld*, *Snyder* u.a. schon angenommen, daß die heterospezifische Schwangerschaft einen Einfluß auf die Entwicklung der Frucht hat. *Koller*, *Haselhorst* u.a. lehnten später eine solche Möglichkeit ab; beobachtete (geringe) Unterschiede führten sie auf Zufall, Unehelichkeit u.a. zurück. Inzwischen hat jedoch die weitere Entwicklung im Grundsätzlichen *Hirszfeld* recht gegeben; ein Neuaufgreifen der Fragestellung ob und inwieweit die gruppenfremde Schwangerschaft merkmaldifferenzierend wirkt, schien so gerechtfertigt. Für eine solche Untersuchung eignet sich das menschliche Hautleistensystem vor allem, da seine endgültige Differenzierung (2.–4. Fetalmonat) zeitlich zusammenfällt mit dem ersten Auftreten der A-B-0 und MN-Systeme.

Tabelle 1. Verteilung des Quantitativen Wertes.

	n	0–2,9	3–5,9	6–8,9	9–11,9	12–14,9	15–17,9	18–20,9	21–23,9	24–26,9
Mütter Gesamt	453	2,2	5,7	11,0	17,0	22,0	22,8	12,9	5,1	1,3
Kinder Gesamt	456	2,1	5,2	9,6	13,1	25,6	23,6	16,1	4,2	0,4
Knaben	227	0,4	4,0	7,0	12,3	27,0	25,5	17,2	6,6	—
Knabenmütter	227	3,2	7,0	11,4	15,8	23,3	21,1	12,3	5,3	0,4
Mädchen	229	3,5	6,5	12,3	14,4	24,0	21,8	14,8	1,7	0,8
Mädchenmütter	226	1,3	4,4	10,6	18,1	20,8	24,3	13,3	4,9	2,2
A-Mütter	226	2,2	6,6	11,1	15,9	23,0	23,0	12,4	4,0	1,8
B-Mütter	56	1,8	5,4	10,7	18,0	19,6	18,0	12,5	14,2	—
0-Mütter	153	2,0	5,2	9,8	19,6	22,2	23,0	13,1	3,9	1,3
A-Kinder	200	1,5	4,5	10,0	12,5	27,0	23,0	19,5	2,0	—
B-Kinder	41	2,4	7,3	9,7	7,3	26,8	29,3	9,7	7,3	—
0-Kinder	205	2,4	5,4	9,3	15,2	24,3	22,9	13,7	5,8	1,0
M-Mütter	120	1,7	5,8	9,2	16,7	25,8	24,2	12,5	2,5	1,7
N-Mütter	89	3,4	3,4	6,8	18,0	22,5	23,5	14,6	4,5	3,4
MN-Mütter	241	2,1	6,6	13,3	17,0	20,0	22,0	12,0	6,6	0,4
M-Kinder	128	1,5	6,3	11,0	13,3	32,8	17,2	16,4	1,5	—
N-Kinder	83	—	2,4	7,2	13,2	32,6	25,3	15,7	3,6	—
MN-Kinder	250	2,8	5,6	10,8	12,0	21,2	25,6	15,6	6,0	0,8
Mutter: Kind N : N	46	—	4,3	2,2	19,6	32,6	26,1	15,2	—	—
Mutter: Kind N : MN	55	1,8	5,5	9,1	18,2	21,8	21,8	7,3	10,9	3,6
Mutter: Kind MN: N	46	—	2,2	15,2	17,4	30,5	19,6	8,7	6,5	—
Mutter: Kind MN: MN	155	3,2	9,1	9,7	9,1	23,2	26,5	16,8	2,6	—
Mutter: Kind MN: M	58	3,4	1,7	8,7	20,7	41,5	13,8	10,4	—	—
Mutter: Kind M : MN	54	3,7	—	20,8	14,8	22,2	24,1	14,8	—	—
Mutter: Kind M : M	76	5,3	9,2	11,8	13,2	29,0	18,4	10,5	2,6	—

Die hier interessierenden Arbeiten über eine Koppelung des Musterbildes an die klassischen Blutgruppen (*Karl*, *Hesch* u.a.) sind in ihren Ergebnissen umstritten (*Geipel*). Über die Beziehungen des Hautleistensystems zu körperlichen und geistigen Eigenschaften berichten *Schlegel*, *Poll*, *Kirchmaier*, *Duis* u.a. *Pich*, *Wendt* und *Zell* konnten eine Korrelation Musterbild–Krankheit nicht bestätigen. Da jedoch Korrelationen zwischen den Musterformen auf den Fingerbeeren und denen der Handflächen bestehen (*Siegle*), der Tastleistenverlauf der Handfläche seinerseits wieder im Zusammenhang mit den Handfurchen und der Handform gesehen werden kann (*Bettmann*, *Schaeuble*, *Würth*), für die konstitutionelle Beziehungen zu Habitus und Krankheitsbereitschaft (*Zenneck*, *Frick*, *Wanner*, *Kühnel*, *Plattner* u.a.) nachgewiesen sind, schien es uns „nicht völlig aussichtslos, auch für die Fingermuster nach gewissen Allgemeinzusammenhängen zu suchen" (*Wünsche*). Die vorliegende Untersuchung schließt sich an eine Reihe von Arbeiten an, die auf Veranlassung von Prof. Dr. Dr. K. Saller mit dem Ziel durchgeführt werden, Zusammenhänge zwischen Hand und Konstitution aufzudecken.

Hier soll geprüft werden, ob Beziehungen zwischen den A-B-0- und den MN-Systemen einerseits und dem Individuellen Quantitativen Wert andererseits bestehen. Zugleich wird damit ein Beitrag gegeben zu der bisher vernachlässigten Statistik des Quantitativen Wertes. In der *Definition* der Blutgruppen und Faktoren halten wir uns an die z.Zt. gebräuchliche Nomenklatur. Der Individuelle Quantitative Wert (i.w. abgekürzt: QW) im Hautleistensystem der Fingerbeeren „ist definiert als der 10. Teil der Summe aus den jeweils größten QW aller zehn Finger. Der Zusatz „größten" ist deshalb zu machen, weil die Wirbelmuster zwei QW haben, von denen nur immer der größere Wert in Betracht gezogen wird" (*Geipel*).

Die *Untersuchung* erfaßt insgesamt 983 Individuen (489 Mütter und 494 Kinder). Die Blutgruppen und Faktoren wurden durch amtliche Gutachter bestimmt; der Prozentsatz der Fehlbestimmungen wird so relativ niedrig sein. Für die statistische Bearbeitung wurde fast ausschließlich das χ^2-Verfahren angewandt.

Verteilung des Individuellen Quantitativen Wertes.

In der *Gesamtverteilung* (Tab. 1) ist der Geschlechtsunterschied bemerkenswert: Er äußert sich in einer größeren Variabilität des weiblichen Geschlechtes gegenüber dem männlichen mit statistisch signifikanten Unterschieden in den Klassen der niedrigen QW. Bei der Aufschlüsselung des *Quantitativen Wertes auf die Blutgruppen des AB0-Systems* (Tab. 1) passen sich die Verteilungskurven der zahlenmäßig größten Gruppen ausgezeichnet aneinander an; sie können als Stichproben aus einer zugrundeliegenden Gesamtverteilung angesehen werden (χ^2-Werte, die einem P zwischen 15 % und 30 % ent-

Tabelle 2. QW bei verschiedenen Gruppen.

Gruppe	Männer		Frauen		Untersucher*)	Bemerkungen
	n	QW	n	QW		
Urker (Holland)	200	13,1	200	12,5	*Piebenga*	1942
Ströbeck	315	13,1	241	12,8	*Karl*	1934
Dithmarscher	83	14,8	90	14,0	*Abel*	
Beitzsch	74	14,6	75	13,1	*Abel*	1938
Schwarzwälder	96	12,2	126	12,4	*Abel*	
Chinesen	70	15,8			*Abel*	
Eskimo	28	20,4	33	20,8	*Abel*	1938
Pygmäen	55	10,1	46	6,9	*Abel*	
Bayern Kinder . .	234	14,1	231	12,5	*Baitsch*	Gesamt
Bayern Mütter . .		—	460	13,1	*Baitsch*	Gesamt
Bayern Mütter . .		—	229	12,7	*Baitsch*	Knabenmütter
Bayern Mütter . .		—	231	13,5	*Baitsch*	Mädchenmütter
Bayern Mütter . .		—	122	13,75	*Baitsch*	Mütter mit Faktor MM
Bayern Mütter . .		—	94	13,70	*Baitsch*	Mütter mit Faktor NN
Bayern Mütter . .		—	241	13,10	*Baitsch*	Mütter mit Faktor MN
Bayern Kinder . .	64	12,8	64	12,3	*Baitsch*	Kinder mit Faktor MM
Bayern Kinder . .	38	15,1	45	12,9	*Baitsch*	Kinder mit Faktor NN
Bayern Kinder . .	132	14,1	118	12,7	*Baitsch*	Kinder mit Faktor MN

*) nach *Fleischhacker*.

sprechen).[1]) Es kann damit für unser Untersuchungsgut als erwiesen gelten, daß eine direkte Abhängigkeit des QW von den Blutgruppen des AB0-Systems nicht besteht.

Bei den *MN-Faktoren* ist die Anpassung weniger gut; die Geschlechtsunterschiede scheinen zurückzutreten gegenüber den Unterschieden, wie sie sich aus der Gruppierung nach den Faktoren ergeben: Knaben und Mädchen mit dem Faktor NN haben schwach besetzte Klassen mit niedrigem QW; bei den MM-Kindern sind diese Klassen stark besetzt, die MN-Kinder halten sich dazwischen. Die Unterschiede sind jedoch statistisch nicht gesichert, so daß eine direkte Korrelation des QW zu den MN-Faktoren in unserem Material nicht nachzuweisen ist.

Der *durchschnittliche QW* (Tab. 2) der Knaben ist mit 14,1 hoch, besonders im Vergleich mit der einzigen süddeutschen Gruppe, den Schwarzwäldern (*Abel*) mit 12,2. Dagegen zeigen die Mädchen (12,5) und die Knabenmütter (12,7) Werte, die den Schwarzwälderinnen (12,4) nahekommen. Der Geschlechtsunterschied ist mit 1,6 stärker ausgeprägt als bei sämtlichen deutschen Vergleichsgruppen. Das vorliegende Untersuchungsgut ist rassisch nicht einheitlich. Es repräsentiert annähernd die derzeitige Bevölkerung Oberbayerns (73 % Einheimische, 27 % Flüchtlinge und Nichtbayern).

Das Mutter-Kind-Verhältnis.

Im Gesamtuntersuchungsgut haben 60 % der Knaben einen höheren und 40 % einen niedrigeren QW als ihre Mütter; von den Mädchen haben 60 % einen niedrigeren und 40 % einen höheren QW. Der Unterschied ist signifikant ($P < 0{,}27\,\%$). – In der Korrelationstafel (Abb. 1) streuen die Mädchen-Mütter-Kombinationen stärker (17 freie Felder) als die Knaben-Mütter-Kombinationen (22 freie Felder). Die größere Variabilität der Mädchen findet sich fast ausschließlich in den Klassen mit niedrigen QW. Die Differenz ist mit einem P zwischen 1 % und 0,27 % statistisch gesichert.

Bei der Verteilung der Differenzwerte auf die *Mutter-Kind-AB0-Kombinationen* (Abb. 2) ist in der Kombination A : A (Mutter A, Kind A) der Geschlechtsunterschied verwischt, das Maximum der Verteilungskurve liegt in der Differenzwertklasse –1; die Verteilungskurve der Kombination 0 : 0 zeigt dagegen eine Rechtsverschiebung. Die Kombinationen 0 : A und A : 0 nehmen eine Zwischenstellung

[1]) Die umfangreichen Berechnungen können im Anthropologischen Institut der Universität München eingesehen werden.

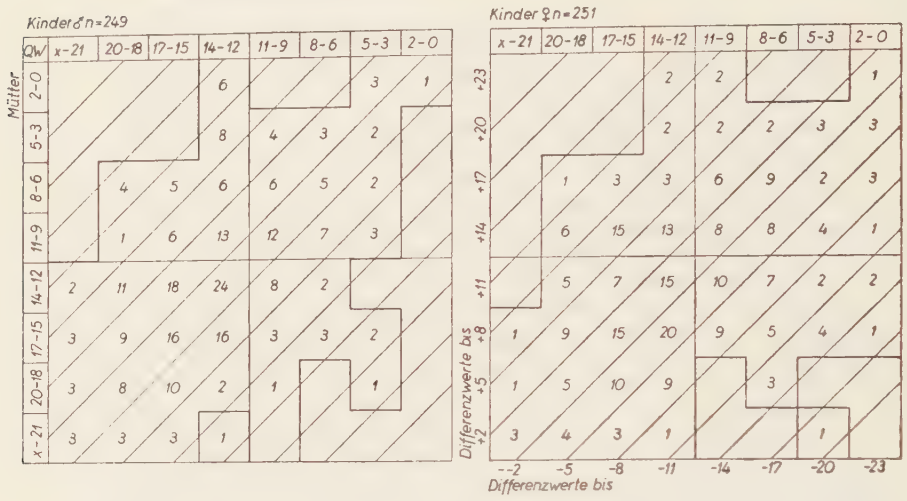

Abb. 1. Korrelationstafel

ein, 0 : A mehr 0 : 0 ähnlich und A : 0 ähnlich A : A. Die Geschlechter verhalten sich in allen Kombinationen gleichsinnig, die Unterschiede sind jedoch bei den Knaben ausgeprägter als bei den Mädchen. Die Unterschiede zwischen der Kombination A : A ♂ einerseits und 0 : 0 ♂ (mit und ohne 0 : A ♂) andererseits lassen sich statistisch schwach sichern (P = 0,6 %).

Je häufiger in einer der *Mutter-Kind-MN*-Kombinationen (Tab. 1 und 3) der Faktor M vertreten ist, desto stärker scheinen die Klassen mit niedrigem QW besetzt, desto niedriger ist im allgemeinen der Durchschnittswert.

Die MM-Kinder der MM-Mütter besitzen einen hohen Prozent-

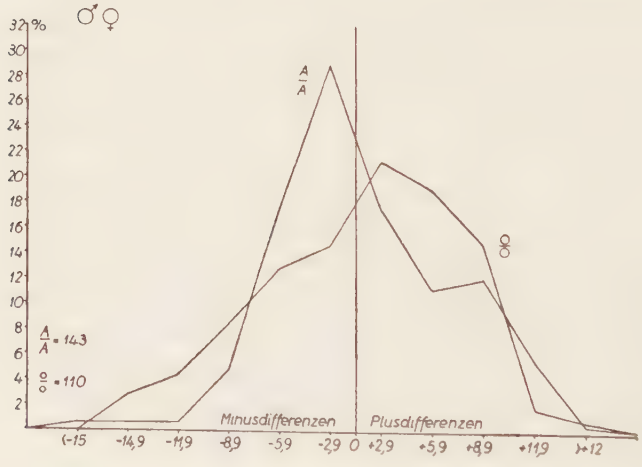

Abb. 2. Verteilung der Differenzwerte.

Tabelle 3. Individueller Quantitativer Wert der Kinder (Mutter-Kind-Verbindungen).

KM : Kind	n	Durchschnittswerte			0,0–8,9	9,0–26,0
NN : NN	46	14,6 ♂	13,1 ♀	14,1 ♂ ♀	3 (6,5 %)	43
NN : MN	55	14,6 ♂	13,6 ♀	13,8 ♂ ♀	9 (16,4 %)	46
MN : NN	46	14,5 ♂	12,4 ♀	13,4 ♂ ♀	8 (17,4 %)	38
MN : MN	155	14,6 ♂	12,2 ♀	13,5 ♂ ♀	34 (22,0 %)	121
MN : MM	58	11,9 ♂	13,4 ♀	12,8 ♂ ♀	8 (13,8 %)	50
MM : MN	54	13,6 ♂	12,0 ♀	12,9 ♂ ♀	13 (24,0 %)	41
MM : MM	77	13,3 ♂	12,0 ♀	12,8 ♂ ♀	20 (26,0 %)	57
A : A	150	14,4 ♂	13,0 ♀	13,7 ♂ ♀	24 (16,0 %)	126
0 : 0	123	13,7 ♂	12,9 ♀	13,3 ♂ ♀	23 (18,7 %)	100
B : B	27			13,5 ♂ ♀	5 (18,5 %)	22
0 : A(B)	51	12,1 ♂	13,6 ♀	12,9 ♂ ♀	10 (19,6 %)	41
A(B) : 0	102	13,8 ♂	12,0 ♀	12,9 ♂ ♀	21 (20,6 %)	81

satz niedriger QW (0–8,9); die NN-Kinder der NN-Mütter haben diese Klasse schwach besetzt. Die Unterschiede sind signifikant (P = ~1 %), wenn man im Vierfelderschema der Gruppe mit niedrigem QW eine Gruppe mit höherem QW gegenüberstellt.

Tabelle 4. QW in den Gruppen-Faktoren-Kombinationen.

	Kinder der Kombination	Individueller Quantitativer Wert				n
		Durchschnitt	0,0–8,9		9,0–26,0	
AB0 isospezifisch	N : N	13,49	2	(6,7)	28 (93,3)	30
	M : MM MN : N	14,67	6	(10,7)	50 (89,3)	56
	MN : MN MN : M – M : MN	13,53	32	(19,3)	134 (80,7)	166
	M : M	12,20	13	(27,7)	34 (72,3)	47
AB0 hetero-spezifisch	N : N	13,47	1	(6,2)	15 (93,8)	16
	N : MN MN : N	12,71	11	(24,5)	34 (75,5)	45
	MN : MN MN : M M : MN	12,49	23	(22,8)	78 (77,2)	101
	M : M	12,39	7	(23,3)	23 (76,7)	30

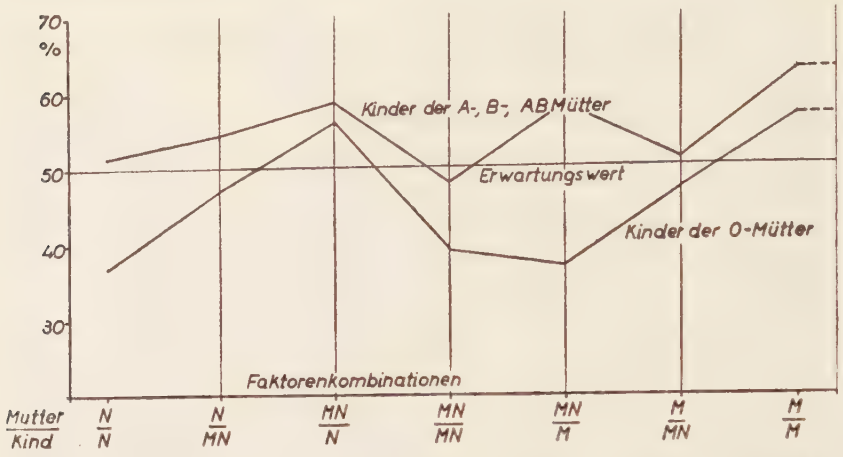

Abb. 3. Häufigkeit der Minusdifferenzen (QW der Kinder niedriger als QW der Mütter).

Bei der Aufgliederung des Gesamtmaterials auf die *Gruppen-Faktoren-Kombinationen* ergibt sich eine schwache Abhängigkeit von der AB0-Spezifität (Tab. 4) zusammen mit dem Vorhandensein des Faktors M, zugleich scheint die Blutgruppenzugehörigkeit der Mutter (Abb. 3) nicht ohne Einfluß zu sein.

Besprechung der Ergebnisse.

Zwei Fragen stehen zur Beantwortung offen: 1. Sind die gefundenen Unterschiede signifikant oder halten sich sich noch im Rahmen des Zufälligen? 2. Wenn die Unterschiede tatsächlich vorhanden sind, wie erklären sich die Zusammenhänge?

Bei der statistischen Bearbeitung gehen wir von der Annahme aus, daß das Elternmaterial hinsichtlich des QW homogen sei. Dies trifft in Wirklichkeit sicher nicht zu; hierzu müßte das Untersuchungsgut um ein Vielfaches vergrößert werden. Da ein solcher Idealfall von einem Einzeluntersucher kaum erreicht werden kann, soll unser kleines Material als Grundlage einer Sammelstatistik dienen, für die meist keine neuen Untersuchungsreihen begonnen werden müßten, da vielerorts entsprechende Unterlagen noch unausgewertet vorliegen. – Eine gewisse Kontrolle über die Zufälligkeit oder statistische Verbindlichkeit der gewonnenen Ergebnisse schien u. a. möglich durch die getrennte Bearbeitung der beiden Geschlechter. Durch die große Zahl der gebildeten Gruppen müßten sich bei Zufallsergebnissen öfters abweichende Beziehungen zeigen. Dies war jedoch nicht der Fall: Sowohl bei den Einzelgruppen als auch bei den nach biologischen Gegebenheiten vorgenommenen Zusammenfassungen waren die gefundenen Beziehungen fast durchwegs gleichsinnig.

Vorausgesetzt, die gefundenen Unterschiede bestehen tatsächlich, wie sind sie dann zu deuten?

Das Eingreifen der Blutgruppensysteme in die Entwicklung des Hautleistensystems könnte über Antigen-Antikörper-Immunreaktionen vor sich gehen, in deren Folge es beim Fetus zu (allergischen?) „Entzündungen" unter aktiver und passiver Beteiligung des Gefäßsystems (*RES*) (*Speiser*, *Schwarz*, *Berndt*) kommt. Sollten diese Vorgänge zu Änderungen der Capillardurchlässigkeit führen, dann ist auch ein Einfluß auf die Fingerbeerenpolster denkbar. (Vgl. die Schleimhautblutungen und das Hydropssyndrom bei den Erythroblastosen sowie die Capillarschädigungen bei den *Little-Bagg*-Mäusen; zur Mechanik der Musterbildung siehe *Bonnevie*, *Abel*).

Da sich in unserem Material auch eine Abhängigkeit von den Blutgruppen der jeweiligen Mütter zeigte, wird man an Vorgänge denken können, wie sie sich bei der parenteralen Einverleibung von fremdem Eiweiß in einem nichtsensibilisierten Organismus abspielen; *Selye* beobachtete (im Tierversuch) dabei an den Akren entzündliche Ödeme. Es scheint nicht ausgeschlossen, daß in der Fetalentwicklung des Menschen ähnliche Reaktionen die Entwicklung des Hautleistensystems beeinflussen. *Hirszfeld* sprach in anderem Zusammenhang vom „ständigen Reiz der Gruppenfremdheit", der vielleicht mit der Fähigkeit zur Ausscheidung von Gruppensubstanz (*Witebsky* und *Mohn*) zusammenhängt.

Es sei betont, daß die vorstehend skizzierten Deutungen nur ein grob schematisierender Versuch sein können. Wir sind uns dessen bewußt, daß die Statistik allein keinen Aufschluß geben kann über die Mechanismen, die modifizierend auf die Musterbildung einwirken. Bevor nicht weitere Beobachtungen an größerem Material vorliegen, erscheint eine ausführliche Diskussion der möglichen Zusammenhänge noch verfrüht.

Inwieweit die Geschlechtsunterschiede im Hautleistensystem, das ein leicht zugänglicher Indikator für tieferliegende Unterschiede zu sein scheint (*Saller*), sich auf „Reaktionsunterschiede" (*E. Fischer*) allgemeiner Art zurückführen lassen, wird z. Zt. experimentell überprüft. Die Besprechung der Geschlechtsunterschiede, die sich bei der vorliegenden Untersuchung ergaben, wird im Zusammenhang mit den experimentellen Ergebnissen nachgeholt werden.

Zusammenfassung.

1. Die Verteilung des Individuellen Quantitativen Wertes im Hautleistensystem der Fingerbeeren (983 Individuen) wird dargestellt; das Untersuchungsgut entspricht nach seiner Herkunft annähernd der derzeitigen Bevölkerung von Oberbayern.

2. Es lassen sich keine *direkten* Beziehungen zwischen dem QW einerseits und den AB0-Blutgruppen und den MN-Faktoren andererseits nachweisen.

3. In Geschlechterzusammenhängen scheint es jedoch je nach der Mutter-Kind-Konstellation zur Modifikation des QW zu kommen.

Résumé.

1. Description de la distribution des valeurs quantitatives individuelles dans les papilles dermiques des bouts des doigts (983

individus). En origine les personnes étudiées correspondent approximativement à la population qui demeurait à cette époque en Oberbayern.

2. Aucun rapport direct entre les valeurs quantitatives individuelles des papilles d'un côté et le groupe sanguin AB0 ou les facteurs MN de l'autre pouvait être démontré.

3. Relativement au sexe certaines modifications des valeurs quantitatives individuelles dans les papilles semblent pourtant exister conformément à la constellation mère-enfant.

Summary.

1. The distribution of the individual quantitative values in the papillary pattern of the finger tips (983 individuals) is described; the investigated individuals correspond in origin approximately to the population in Oberbayern at that time.

2. Any direct connection between the individual quantitative values in the papillary pattern on one hand and the AB0 blood group or the MN factors on the other could not be shown.

3. However, certain modifications of the individual quantitative values in the papillary pattern seem to occur in connection with the sex according to the mother-child constellation.

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Tage Kemp: **Genetics and Disease.** Munksgaard, Copenhagen 1951, 330 pp.

This book written by the distinguished leader of the Institute for Human Genetics in Copenhagen should be very welcome to everybody teaching a genetic course to medical students. So far there has been very few books[1]) which were written by physicians specialized in medical genetics *for* physicians or medical students. Especially one would like to emphasize that the excellent cooperation between clinicians and geneticists in research, as well as the accumulated experience in handling counseling and other practical applications of medical genetics, at the Danish Institute is a guarantee that this book foots on firm grounds. It contains solid information on many genetic questions which might meet the physician in his clinic or office. It also gives a comprehensive summary of modern genetic hygiene indispensable for the Public Health Officer.

The text can be fully utilized without any previous knowledge of genetics. Part I explains the basic principles of genetics stressing facts of immediate interest for the understanding of human genetics. Part II is devoted to general principles of medical genetics, part III to the inheritance of normal traits. The genetic diseases of different organ systems are reviewed in part IV. Here one finds much valuable information about frequencies of different genetic disorders, morbid risks and, as far as we know it at present, about the mode of inheritance. Part V, finally, explains the principles of genetic hygiene. The adoption of the term genetic hygiene to substitute eugenics is very fortunate. It stresses the fact that the practical applications of medical genetics is strictly a medical and public health business. The immature eugenics movements, books and pamphlets which were widespread in the twenties and thirties and raised to a crescendo in the Third Reich until the balloon cracked did much to discredit medical genetics. Many physicians are still reluctant to genetics because they have not realized that these ideas since long have been abandoned by students of human genetics.

The use of genetic principles and methods in medical research is becoming increasingly important. Patients with "constitutional" disorders accumulate in the clinics and something more should be done about it than giving these patients a diagnosis and at the best a symptomatic treatment. It is apparent to the specialist

[1]) It may be mentioned that already in 1934 Professor *Otto L. Mohr* published his book "Heredity and disease" and that Professor *Gunnar Dahlberg* in 1942 published his book "Race, Reason and Rubbish" which was also published in the Danish, Finnish, German, Norwegian and Swedish languages.

in medical genetics that pathogenetic genes constitute as important causes of human disease as do bacteria and viruses. But how many medical schools give courses in medical genetics? Dr. Kemp's textbook gives the necessary background for an understanding of the fact that genetics can no longer be considered a negligible part of medicine. One might therefore hope that the book will stimulate interest and give impulses to courses in medical genetics at the medical schools. This seems the best way to stimulate future research on the accumulated "constitutional" disorders on a broad basis.

Naturally if a textbook is scrutinized one will find some errors and inconsistencies. On a few points one is inclined to disagree with the opinions expressed. The chapter on methods of medical genetics is somewhat meager, but on the other hand the book is not intended as a text for research workers. A few beauty spots, however, should be mentioned for consideration to the next edition. As concerns the sib method (p. 124) it should have been clearly expressed that this calculation can be used if *all* character-bearers have been ascertained without selection, only. This, for instance, will be the case if all cases with a certain disorder have been registered as propositi in a given population. The same requirements must be fullfilled if one wants to use the *a priori* method of Bernstein. It is not quite clear if "the *a priori* propositus method" mentioned on p. 125 refers to the latter. If so it is incorrectly stated that "the propositi themselves are not included in the counts". The *a priori* method starts with a theoretical ratio of 1 : 1, 1 : 3 or whatever one might choose. By binomial expansion the expected distribution of affected and unaffected in sibships of different sizes is calculated. By exclusion of those sibships which contain unaffected members, only, which cannot be ascertained, one calculates the expected ratio of affected : unaffected. These expected ratios include all affected members and thus no exclusion of propositi should be made in contrast with the methods of Weinberg. Only one-child-families can be excluded as here the expected and observed ratio by definition will always be the same, namely 100 per cent.

On p. 131 the formula for the probability that two egg twin pairs in which one twin has the disease show concordance is given as

$$\frac{100\ p}{2 - p}$$

where p is the probability that a genetic disease might occur in a sibship. This does not seem correct and the derivation of the formula is not given. As far as the writer can see the probability of concordance is simply p (= the expected incidence among ordinary sibs). Suppose we are dealing with a simple dominant with complete penetrance, one parent always being likewise affected. The expected frequency of affected children (or sibs) is 50 ($p = \frac{1}{2}$) per cent. If we collect n affected dizygotic twin partners $\frac{1}{2}$ n of their twin siblings will be expected to be affected and consequently 50 per cent of the twin pairs will show concordance. On the other hand, if we collect *all twin pairs* from marriages in which one parent is affected the expected incidence of positively concordant pairs will be p^2 or in the present example 25 per cent.

These are small disturbing elements in an otherwise excellent text. Dr. Kemp's book, on the reasons mentioned previously, is recommended as furnishing interesting and indispensable information on genetics in relation to theoretical and clinical medicine to medical students, clinicians of different specialities, students of public health and social workers.

Jan A. Böök, Uppsala

Karlheinz Idelberger: **Die Erbpathologie der sogenannten angeborenen Hüftverrenkung** (The genetics of congenital dislocation of the hip). Urban und Schwarzenberg, München and Berlin 1951. 80 pages. RM 11.–.

The author who is a specialist in orthopedic surgery should by now be known by every human geneticist and orthopedician. By that I am referring to Dr. *Idelberger's* exhaustive twin study of congenital clubfoot (Ztschr. f. Orthop. 69, 1939). This is so far one of the largest single contributions to the genetics of a specific clinical condition. It cleaned up a lot of wishful thinking and expelled (or should have) to the land of fairy tales some of the loved mechanical speculations about the etiology. To sum up, the *basic* genetic origin of the vast number of club feet was convincingly demonstrated.

This new study concerns congenital dislocation of the hip which occurs with an average frequency of 1 : 1.500 newborns (the incidence being 5 to 8 times higher in females than in males). Previous studies of pedigrees by *Faber* and others in Germany have clearly indicated the genetic etiology of this defect but sufficient twin data have been lacking.

Dr. *Idelberger* collected 22.004 cases from all orthopedic and surgical clinics together with cases reported to the Cripples Aid Organization in Germany during 1921–39. Of these 236 were twins. For one reason or the other, one or both twins deceased, incorrect diagnosis etc., 98 pairs were excluded. The remaining 138 pairs, consisting of 29 identical, 52 like-sexed and 57 unlike-sexed fraternal pairs, were examined. The incidence of twins in the total material is significantly lower than expected (1.07 per cent against an expectation of 1.8 per cent) which constitutes a serious argument against the idea of a mechanical factor operating *in utero*. The analysis of maternal age, distribution on different birth ranks, maternal condition during pregnancy and obstetric history did not reveal anything of significance. The author states that he is unable to find any consistent or significant environmental cause. The unability to find an exogeneous etiology, of course, does not prove that such causes once and for all have been excluded even if the work as in Dr. *Idelberger's* case has been carefully done. However, it offers a legitimate reason to explore the possibilities for a genetical explanation.

As the manifestation of a congenital hip has been discussed in terms of different degrees of acetabular pathology, varying from slight changes detectable by X-ray, only, to the full-blown clinical condition, some standards have to be used when the twin siblings are compared. Dr. *Idelberger* used two different levels of concordance, namely (1) both partners display the wellknown clinical type and (2) one partner, the propositus, displays the full-blown type (this by the way always being the case as the *propositi* were selected according to this principle) and the other one displays an X-ray detectable dysplasia of the acetabulum. Of the 29 monozygotic pairs 10 displayed concordance according to (1) above and an additional two pair concordance at level (2), in other words a total concordance of 41.4 per cent. In contrast to this the 109 dizygotic pairs displayed a concordance of 2.8 per cent, only [all at level (1)]. The difference is statistically highly significant. Furthermore the 2.8 per cent affected dizygotic twin siblings agree with the incidence of affected among 4.886 sibs of propositi with congenital hip (also 2.8 per cent) as reported previously by Dr. Isigkeit.

It is of special importance that concordance at level (2) appears rather insignificant as the reports by Dr. *Faber* indicated that X-ray detectable acetabular dysplasia should be very common among the relatives of the propositi. He also offered a hypothesis of simple dominant transmission provided one added together the dysplasias and full-blown cases in the families. Very likely Dr. *Faber*'s pedigrees are in some way or the other biased or represent exceptional cases and should not be generalized.

All concordant pairs were like-sexed and female. This makes the penetrance figure, calculated at 60 per cent, valid for this sex, only. The dislocation was bilateral in 41 per cent of the cases which agrees with the concordance figure for the monozygotic twins. This might be taken as another indication of the basically endogenous origin of the condition.

Dr. *Idelberger*'s data and the analysis thereof give very good evidence in favor of the theory that most cases of congenital dislocation of the hip develop on the basis of a specific genotype which, however, appears relatively plastic in its response to the environment. This is important information. Naturally many questionmarks remain as those of the genetic homogeneity of the data, the specific action of the gene or genes involved, the specific action of environmental stimuli which are capable of inhibiting gene action etc. Further research in this field cannot afford to neglect this careful study. Jan A. Böök, Uppsala.

M. Murphy: **Heredity in Uterine Cancer.** Harvard University Press, Cambridge, Mass., 1952. 128 pages. $ 2.50.

This study which was planned on similar principles as used by Brøbeck (Heredity in Cancer Uteri. Aarhus, Denmark, Universitetsforlaget 1949) deals with 201 propositi with cancer of the cervix uteri verified by microscopic diagnosis and 215 control propositi. The occurrence of uterine cancer as well as cancer of other sites in mothers, sisters, female cousins, aunts and mother's and father's brother's wifes was studied. One wonders why the last two categories were included as "relations" of the propositi. The data include information about 2,809 relatives of the cancer propositi and 3,220 relatives of the control propositi. As a rule the propositi had reached an age of 45 years. It would seem that cancer of the uterus (cervix and corpus) occurs with somewath increased frequency in the mothers of the cancer propositi though the significance is at the level of $P = 0.02$, only. For sisters no significant differences were found. As far as I can see this remains the essential result of this study. In as much as the incidence of uterine cancer and cancer of other sites in the different degrees of relations is concerned one does not feel convinced that they represent anything of significance.

One might raise a number of objections against this study, some of them rather serious. Personally I do not believe that, as far as specific diseases are concerned, medical genetics as a rule can be much furthered by studies based on questionnaires filled out by non-professional people, no matter how experienced the composer of the questionnaire. At the most such a procedure might serve as a pilot study. It appears from the book that the author did not perform any actual medical examinations of the individuals included. If one would remark that the number is too large for such a venture one would wonder if anyone really thinks that large numbers will compensate for superficiality. One cannot help asking what would

have been the result if for instance the sisters of the cancer propositi had been subjected to a gynaecological examination (and of course likewise the sisters of the control propositi). If genetics is important in regard to uterine cancer how does one know that a supposed genetic type of uterine cancer shows the same symptomatology as uterine cancer in randomly selected individuals? It could grow more slowly and remain unsymptomatic for such a long time that an appreciable number of individuals would die before having a chance to be detected. By using such a superficial procedure one puts so many, I should say unnecessary, unknowns in the equation that one actually runs the risk that the questionmark becomes larger at the end than it was at the beginning.

In as much as we are concerned with genetic or supposedly genetic disorders which occur with a high incidence in the population (i.e. at the 1 % or higher level) it does not pay to include other individuals in the study than parents, sibs and children. The risk figures (or incidences) for more distant relatives will come too close to the general incidence in the population so that extremely large bodies of data will be necessary if one wants statistically significant differences. Under such circumstances one must urge that first and foremost the relations mentioned above be thoroughly investigated. It also appears from Dr. *Murphy's* data that the information obtained about more distant relatives who constitute the majority of the individuals is practically nil.

The statistical analysis is based on simple comparisons as the data were collected as to correspond in a number of relevant attributes as age, social conditions etc. This is hardly sufficient as concerns cancer. It seems necessary to calculate morbidity risks and mortality risks constructed for comparisons with the control data and with the random risk.

As well acquainted with the difficulties involved in the study of family groups in the United States and granting that the topic of heredity in cancer is a knotty problem the reviewer appreciates that a trial has been made to collect information about the families of propositi with uterine cancer. Another thing is that the reviewer must regret that he could not find that anything essentially new came out of it and that the conclusions will remain debatable. Jan A. Böök, Uppsala.

ERRATA

Through an unfortunate oversight the bibliography was left out in *E. Otterström*'s paper "The Social Outlook for Children of Divorcees", published in fasc. 1, vol. 3 of this journal. The literature cited are as follows:

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From the State Institute of Human Genetics and Race Biology, Uppsala, Sweden
(Head: Professor Gunnar Dahlberg, M. D., LL. D.)

Tooth Size and Occlusion in Twins

by Anders Lundström

206 pages with 29 figures. 1948. Price sFr. 16.65

Out of the contents:

Review of the Literature. Methods of Investigation. Statistical Methods Used. Cases Investigated. The Cases from a Representative Point of View. Loss of Teeth in Twins, and their Importance to the Material from a Representative Viewpoint. The Variation in Twins regarding Breadth of Teeth and Occlusion. Cases of Extreme Malocclusions. Significance of Genetic and Non-genetic Factors as regards the Tooth-Breadth and the Occlusion.

From the State Institute of Human Genetics and Race Biology, Uppsala, Sweden
(Head: Professor Gunnar Dahlberg, M. D., LL. D.)

Mathematical Methods for Population Genetics

by Gunnar Dahlberg

182 pages with 32 figures and 31 tables. 1947. Price sFr. 26.–

Out of the contents:

1. Division of characteristics from a hereditary viewpoint. 2. The conception of race and the laws of Mendel. 3. Different forms of inheritance. 4. Composition of populations in panmixia, 5. The effect of mutations on the composition of a population in panmixia. 6. The effect of selection on a population. 7. Selection and mutations. 8. The effect on inmarriage on a population. 9. Inmarriage and selection. 10. Assortative mating. 11. Assortative mating, inmarriage, selection, and mutations. 12. The importance of the isolate for the composition of populations. 13. Isolates and race. 14. Mutations, selection, and isolates. 15. Isolates and inmarriage. 16. Isolates, assortative mating, and selection. 17. General survey.

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